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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

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Original Papers.

A FORM OF PROGRESSIVE CEREBRAL SCLEROSIS IN INFANTS ASSOCIATED WITH PRIMARY DEGENERATION OF THE INTERFASCICULAR GLIA.

By

J. G. GREENFIELD, London.

Progressive cerebral sclerosis in young children assumes two main forms for which the generic names of amaurotic family idiocy and progressive centrolobar sclerosis are perhaps the most suitable. The first has during the past 20 years largely yielded up its secrets to the histological investigations of a number of workers. The studies on neuronal metabolism in this disease by Marinesco, and of its Mendelian characteristics by Sjögren, deserve special mention in this connexion. Of the second form we know less, and the theories which have been advanced to account for it are largely speculative. Bielschowsky and Henneberg have divided cases of this kind into two main groups, the non-familial type of Schilder which they assume to be due to an exogenous infection, and a familial type which is attributed to an endogenous degenerative process. The latter has again been divided according to age-incidence into the infantile type of Krabbe, the juvenile type of Scholz and the slowly progressive type of Pelizaeus and Merzbacher. This division is somewhat arbitrary, since there is increasing evidence that many of the cases which have been described as Schilder's ' encephalitis periaxialis diffusa ' are familial. Thus Collier and Greenfield's case II appeared at first to be isolated, but three younger children of the family have since become affected on reaching a similar age; the post-mortem examination of the first two of these has revealed lesions in the brain which are almost identical with those described in the first child. It is noteworthy that in most families, though not in that of Symonds, the disease appears at the same age and runs a similar course in each of the members affected; it is no longer possible to describe a few main types, according to age of incidence and rapidity of involvement of the cerebral functions, seeing that the type varies with each affected family.

From these familial cases which run a subacute course the form described by Pelizaeus and Merzbacher is fairly sharply delimited, owing to the extremely slow course of the disease and the peculiarly patchy destruction of the central white matter of the brain. I have had no opportunity of examining a case of this kind, and shall therefore confine discussion to subacute cases.

According to Bielschowsky and Henneberg the true cases of Schilder's encephalitis, that is those due to an exogenous infection, are differentiated from the familial degenerative cases by the presence of lymphocytic exudate round the vessels in the recently affected areas, and by the tendency of demyelinization to begin as perivascular zones which coalesce as they enlarge. (The very rapidly fatal case in an adult recorded by Stewart, Greenfield and Blandy in 1927 was of this nature.) In the degenerative and often familial type, they note that the process of myelin disintegration assumes a different form from that found either in Wallerian degeneration or in such acute demyelinating processes as disseminated sclerosis. Thus atypical pre-lipoids are formed which resemble in some ways those which fill the nerve-cells in amaurotic family idiocy, a point which has also been made by Coenen and Mir. They suggest that the origin of the trouble may lie in the circulation of abnormal lipoids in the blood, which are unsuitable for the formation or nutrition of myelin.

Collier and Greenfield (1924) suggested that Schilder's disease, in the familial case which they studied, was due to a primary affection of those neuroglial cells in the white matter which had to do with the nutrition of the myelin sheaths. The nutritive function of the oligodendroglia towards myelin was not recognised at this time, and in fact the first English description of oligodendroglia appeared in the same number of *Brain*.

Scholz considered the disease in his family to be a hereditary anomaly of the power of the neuroglia to regulate the metabolism of myelin. But he also pointed out that the myelin sheaths were most damaged in the regions of poorest blood supply.

Levaditi has recently propounded a somewhat different theory to account for the demyelination. He considers that it is due to a disturbance of lipoid metabolism on the part of the oligodendrocytes, so that instead of nourishing the myelin sheaths they remove the myelin from them. This inversion of the normal function of the oligodendrocytes is attributed to the direct action on them of a neurotropic virus which he terms ' oligodendrophile.'

Numerous workers, especially in America, have emphasized the fact that the oligodendrocytes tend to degenerate in the affected zones, usually going through the stage of acute swelling, and in many cases forming mucinoid material which with the disintegration of the cell-body escapes into the tissues. Eventually oligodendrocytes may completely disappear from the central parts of the degenerated areas; but, as far as can be gathered

from descriptions and microphotographs, oligodendrocytes are always present in the less affected zones near the cortex.

It has occurred to some observers that the degeneration of oligodendrocytes may be independent of the demyelination, and may even be primary. Thus Del Rio Hortega (1927), being struck with the simultaneous disappearance of myelin and oligodendrocytes, was led to make the tentative hypothesis that oligodendrocytes might not only act as isolating and supporting structures but might also have to do with the nutrition of the myelin sheaths or the elaboration of myelin. This hypothesis has received some support from the work of Berliner, who observed that oligodendrocytes were present in the retina of the rabbit only in the areas where the nerve-fibres were myelinated. He suggested that the absence of a lamina cribosa in the rabbit permitted oligodendroblasts to migrate into the retina near the disc, and that they then formed myelin round the retinal nerve-fibres. These observations require corroboration. Before we can deduce from them a definitely myelogenetic function for the oligodendroglia, we must know whether opaque nerve-fibres can be present without oligodendrocytes, and vice versa. That the interfascicular glia has some nutritive action on the myelin sheath is however very generally accepted, and its homology with the cells of Schwann has been emphasized both by Del Rio Hortega and by others.

The two cases of progressive cerebral sclerosis here recorded, although they appear to differ in several respects from any cases of Schilder's disease which have been hitherto described, indicate the close association between demyelination and degeneration of the interfascicular oligodendrocytes. They are singular in that the disease has affected tracts and fibre-systems rather than contiguous areas, and in that the oligodendroglia has degenerated and disappeared from fibre-systems which are still fairly well myelinated.

PERSONAL CASES.

Case I. B. R. (female), age 3, was admitted to the National Hospital, Queen Square, under Dr. Critchley, on January 12, 1932, and died on January 16. She was the only child of healthy parents, born at full term and weighing then 7½ lbs. Pregnancy had been normal except that the mother had felt no movements until one week before her confinement. Labour was normal. The child had been lively and vigorous as an infant. She walked when 11 months old and talked at 16 months. When 18 months old she developed a squint which her mother attributed to a fall on her forehead. This was cured by glasses in six months. At the age of 22 months she had whooping-cough. At the age of two she began to fall backwards when walking, at first only two or three times a day, but progressively more often, so that in six weeks she could only walk when she held her mother's hand. By the time she was two and a quarter she could only stand when holding a chair, and when she was two and a half she could do nothing but lie and had lost the use of her hands. The mother stated that there had been no staggering or unsteadiness, but simply progressive loss of strength. The child had also complained of a sensation of

pins and needles at first in her legs and later in her arms, which lasted for three months from the age of two and a half years. At this time she was quite bright, could see and speak normally and could move her head. She was sent first to an orthopædic hospital in Birmingham and after a few days transferred to Leicester Royal Infirmary. When there she changed rapidly, becoming speechless and senseless; she did not know her mother, but turned her head continually from side to side as though it hurt her. After about eight weeks she appeared rather brighter, so that she knew her mother and father, but she seemed to have pain in her limbs. She appeared to be able to see at this time. During the last three months before admission to the National Hospital her eyesight had failed gradually and for the last four weeks she had been blind. She was still able to swallow but did not chew her food. There had been no convulsions at any time.

On examination in the National Hospital she was seen to be a fairly well nourished child of the normal size for her age. She lay in bed with the head retracted, the arms extended, the back bent forwards and the legs crossed. She whined continually. She was quite incooperative and inattentive, but she opened her eyes widely if her name was spoken in either ear. She seemed to be quite blind. The skull measured 49·5 cm. in its greatest circumference. The pupils reacted to light but constantly varied in size. The optic discs and retinæ appeared normal. The corneal reflexes were absent, but pinprick was responded to over the face and body by increased crying. The limbs tended to be held rigidly, especially if handled. The arms were held extended at the elbow, pronated with the fingers tightly closed. The legs were somewhat flexed at the hip, and extended at the knee, the left being crossed over the right. The feet were inverted. Passive movements of the head caused alterations in the position of the limbs to some extent. The deep and superficial reflexes were completely suppressed with the exception of the plantars. Here stimulation of the soles of the feet produced fanning of the tones on both sides, with little dorsiflexion of the great toe.

She died from bronchitis on the fourth day after admission to the hospital.

PATHOLOGY.

The post-mortem examination was delayed for 44 hours after death and the brain only was examined. The dura mater was slightly adherent to the skull. The brain appeared congested but otherwise its external surface looked normal. It was soft but not abnormally so. The occipital convolutions were small and numerous, but there was no obvious shrinkage of this part of the brain. The corpus callosum tore easily. On making horizontal sections through the brain the white matter appeared rather greyer than normal and the centrum ovale was much narrower than in Case II. In the occipital lobes the subcortical fibres looked rather whiter than the deeper zones of the centrum ovale, but the difference was slight. The optic thalamus was shrunken, pale and firm, and the internal capsule was softened in its inner zone and had split away from the optic thalamus and the body of the caudate nucleus. The island of Reil was sunk rather deep beneath its opercula, apparently owing to the shrinkage of the optic thalamus.

Sections stained for myelin by the Wolter (Kulschitsky-Pal) method showed a widespread deficiency of myelinated fibres, most evident in the

more posterior (occipital) parts of the white matter, while the optic radiations, which were relatively intact, showed up as a dark band. There was however some loss here also and many of the myelin sheaths were beaded or locally swollen. In the frontal lobes many fine myelin sheaths were visible under the microscope, although the general appearance to the naked eye was that of complete demyelination. There was no sparing of the subcortical zone, but some myelinated fibres were present in the white centre of a few of the convolutions, and radial fibres and a few tangential fibres were seen

FIG. 1. Horizontal section of the occipital lobe, stained by Weigert-Pal method, showing the general absence of myelinated fibres except in the optic radiations, which stain fairly dark.

in the deeper layers of the cortex in many places. The internal capsule was completely demyelinated in its anterior limb and very pale in the anterior third of the posterior limb, but some rather thin fibres ran vertically in the posterior limb. Some of these were probably afferents from the thalamus. The fibres passing across the internal capsule to the putamen and globus pallidus on the other hand were quite darkly stained. The fibres in the outer part of the thalamus and on its inner border were also well-stained, but

there was general pallor of the internal nucleus of the thalamus. In the brainstem all the tracts with the exception of the pes pedunculi and pyramidal tracts were well stained. In the pes the pyramidal tract appeared as a pale grey wedge-shaped area in the central half; this contained numerous fine vertical fibres. In the inner and outer quarters of the pes no fibres were seen except those which passed across it from the basal ganglia. In the lower part of the pons and in the medulla the pyramidal tracts contained fairly numerous rather thicker fibres, many of which had an oblique course, apparently passing dorsally as they descended. The myelin sheaths in the pyramidal tracts were definitely thicker in the medulla than in the midbrain and internal capsule. The optic nerves stained somewhat palely, but under

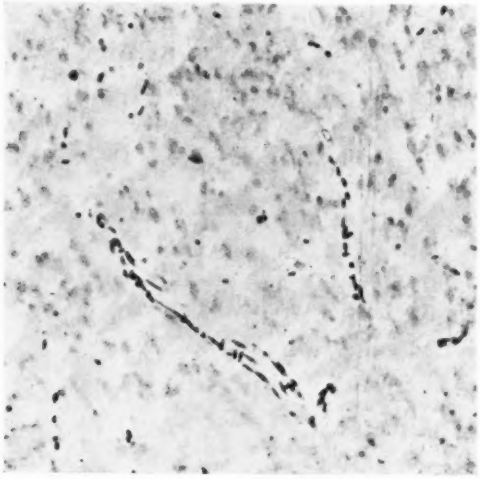

FIG. 2.—White matter of centrum ovale. The only nuclei seen are those round small vessels and isolated nuclei of astrocytes and microglia. Masses of lipoid and mucinoid material are faintly stained. Iron-hæmatoxylin, van Gieson (celloidin section).

the microscope were seen to contain many fairly thick myelin sheaths, many of which were swollen locally with balls of myelin.

In the cerebellum there was complete demyelination of the white matter both in the centre and in the folia over an area corresponding roughly to the lower half.

In sections stained for neuroglia by Mallory's phosphotungstic acid-hæmatoxylin and by Anderson's Victoria blue method a great increase in the number of fibres was found, which resulted in a dense felting in the white matter of the hemispheres and in a general isomorphic increase of

fibres in the brainstem. None was seen in the cerebral cortex or the lenticular and caudate nuclei, except just under the pial surface and the ventricular wall, but in the molecular layer of the cerebellum in its lower half the radial fibres were more distinct and thicker than normal. This neuroglial increase was thus seen not only in demyelinated areas but also in less intense form in parts where the myelin picture was perfectly normal. It was however noteworthy that whereas in the myelinated areas the neuroglial fibres were fine and lay in their normal arrangement, in the non-myelinated areas large cells with swollen cell bodies and thick processes were abundant and a few monster cells were also seen.

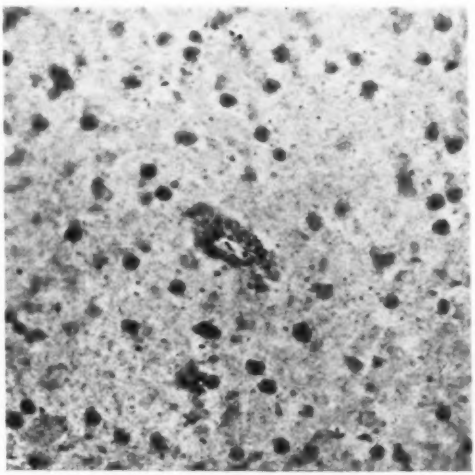

FIG. 8. White matter of centrum ovale, stained by mucicarmine and alum-hæmatoxylin. The mucinoid masses which stain fairly deeply are scattered through the tissues and distend the sheath of a small vessel. The lipoid masses do not stain. Note that the great majority of the mucinoid masses are devoid of nuclei.

In sections stained with hæmatoxylin two striking abnormalities were noted. The first was the complete absence of the small round nuclei of the interfascicular glia cells from large areas of the brain. In fact no cells of this type could be identified with certainty. In the centrum ovale no nuclei could be seen except sparse pale oval nuclei of astrocytes, and irregularly-shaped microglial and endothelial nuclei round the vessels. A similarly deserted appearance was noted in the white matter of the cerebellum. This abnormality was not confined to the demyelinated areas, for the myelinated bundles traversing the globus pallidus, in which interfascicular glial nuclei

are normally abundant, were completely devoid of such nuclei; nor were any seen in the hilum of the inferior olives, although the fibres here also were fairly well myelinated.

The other striking feature was the presence of rounded or oval masses about 20 μ in diameter which stained fairly darkly with hæmatoxylin in frozen and celloidin sections. These were thickly scattered in the demyelinated areas, where they also distended the Virchow-Robin spaces of many of the vessels. They were most abundant, and stained most deeply in a zone which lay about 2 mm. from the cortex. The majority were completely devoid of nuclei, but in a few a minute granule which stained deeply with hæmatoxylin was seen. This may have represented a pyknotic or degenerated nucleus. With mucicarmine and metanil yellow the majority stained rather bright red, and with Heidenhain's Azan stain and Mallory's connective-tissue stain, stained fairly darkly with anilin blue, indicating their mucinoid character. But with the Azan stain there were also numerous similar masses which stained bright red, and in some both blue and red granules were seen. It was noted that in a zone a millimetre wide under the cortex the majority of the masses was of these two types, whereas internal to this zone the great majority of the masses contained only blue-staining granules. Although rounded in outline they did not appear to be enclosed in a definite cell-membrane. These masses were seen not only in the white matter of the cerebral and cerebellar hemispheres but also in the demyelinated areas of the thalamus and among the thalamo-striate fibres. They were visible in the pyramidal tracts in the internal capsule, midbrain, pons and medulla, although at the lower levels they were of smaller size. In sections stained by the Bielschowsky method, the network of axis-cylinders in the frontal region was relatively intact. Only here and there was a local globular axonal swelling seen. In the occipital lobes, there was rather more degeneration of axis-cylinders, so that in some places the network was somewhat wide-meshed, and local bulbar swellings on the nerve-fibres were more numerous. But even here there was a marked contrast between the degree of demyelination and the destruction of axis-cylinders.

Case II. A. H. (male), age 3, was admitted to the National Hospital in January, 1932, under Dr. Collier.

His mother and father were healthy and were not blood relations. They had had four children. The first died at the age of two years and eleven months. He was a boy born at full term; he appeared normal at birth and sat up at 10 months but never walked or talked. From the time of his birth his head was large and gradually increased in size. He was seen at the National Hospital in 1923 and the diagnosis then made was that of hydrocephalus. He had no fits and was not blind.

The second child was a boy of ten and the third a girl of seven. Both of these were kindly examined for me by Dr. Carmichael in the out-patient department and found to be perfectly normal, healthy children. The head circumference of the elder was 21 inches and of the younger 20½ inches.

The patient was a full-time child. The birth was normal. He was breast fed for 10 months. He sat up and talked at the normal ages but had never walked. At the age of 12 months he had measles and was ill with bronchitis after this for three or four weeks, but recovered and at the age of 18 months could roll and crawl about the floor. When he tried to walk his legs became crossed. Otherwise he remained in good health till three months before admission to hospital, i.e., at the age of two years and nine months, when he became ill with influenza. He ate very little, slept and dozed most of the time and his head seemed heavy. Since then he had never sat up. Two weeks later his speech became indistinct, and after a further two weeks he ceased talking altogether. For the last six weeks his mother had noticed that when he cried he became stiff; but there was no loss of consciousness or fits at any time, nor any vomiting. The child ate and slept well but had been increasingly irritable. The mother thought that he was still bright and intelligent and that his sight and hearing were unaffected. His head was said to have been of normal size at birth but to have grown large gradually.

He was found on examination to be a thin small child with a large head. He lay quietly on his back but every 10 minutes or so he appeared to have an attack of pain, his back became arched and his head retracted and he gave a sharp cry. At first all his limbs were rigidly extended, but soon the left arm was flexed and drawn over to the right shoulder. The examination of his eyes showed that the pupils reacted slowly to light. The fundus oculi was normal except for slight fullness of the retinal veins. The corneal reflexes were present, but he did not respond to pinpricks on the face, trunk or limbs. His limbs were held rigid, the left arm and the legs extended and the right arm flexed at the elbow and fingers. The tendon jerks were present but rather feeble, the abdominal reflexes were absent and the plantars of the Babinski type.

A lumbar puncture was performed on January 7. The cerebrospinal fluid was under low pressure (96 mm.). The fluid was clear and colourless. It contained 120 mm. of protein per 100 c.c. and gave a positive Pandy and a weakly positive Nonne-Apelt reaction. No cells were seen. He died the same evening.

PATHOLOGY.

A post-mortem examination was made by me on the following day. There was comparatively little thinning of the skull; the membranes covering the brain were thin and the foramina of Magendie and Luschka open. There was no evidence of hydrocephalus or of thrombosis of the venous sinuses. The convolutions of the brain were rather more prominent than normal and the cerebral hemispheres felt very firm, almost hard, whereas the pons and cerebellum had the normal consistency. The spinal cord was removed. It appeared normal. The left lung was adherent to the left side of the chest and to the diaphragm. Otherwise the lungs appeared normal. The other organs of the body showed no abnormality. The posterior half of each eye was removed, 20 per cent. formalin having been injected into each overnight. The brain weighed 1,287 grammes; the right hemisphere 580 grm., the left 575 grm., and the brainstem and cerebellum 132 grammes. The relation of

the weight of the cerebral hemispheres to that of the brainstem and cerebellum was therefore as 8·75 is to 1, the normal, according to Dunlap, being in the adult as 7 to 1.

The microscopic appearances in this case were so similar to those in Case I that it is only necessary to indicate the details in which they differed. In this case the degeneration was less advanced, in that throughout the white matter of the hemispheres there was a network of fine myelinated fibres interspersed with many myelin balls. Thus to the naked eye the sections appeared pale grey. On the whole there was more demyelination in the occipital than in the frontal lobes, but the difference was not great. The optic radiations stood out as a more darkly stained band, but here also some myelin balls could be seen under the microscope. The appearances of the basal ganglia were the same, except that there was less shrinkage and demyelination of the inner nucleus of the thalamus. In the midbrain the appearance was almost exactly similar to that in Case I, in that while the pyramidal tracts stained faintly the frontopontine and occipital fibres in the crura were unstained. In the cerebellum all the white centre, including the stems of the folia, was fairly well myelinated, although it stained less deeply in the lower than in the upper half. In the cord the pyramidal tracts were pale and the sides of the cord were shrunken in this region, but otherwise there was no abnormality. The optic nerves as a whole stained fairly dark, but on microscopic examination the fibres were rather isolated from one another and irregular in calibre, and balls of myelin could be seen here also. With hæmatoxylin alone similar masses of what appeared to be lipoid material could be seen. This certainly stained less intensely after 48 hours in such fat solvents as xylol, chloroform and a mixture of absolute alcohol and ether, but it did not disappear completely. In sections stained with Scharlach R and alum-hæmatoxylin many of the vessels in the white matter of the cerebral and cerebellar hemispheres were ringed with a layer of granule-cells with bright red fatty inclusions, but no brightly staining lipoid could be seen outside the perivascular sheaths. The dark masses which were seen in sections stained with iron-hæmatoxylin only, appeared after Scharlach staining as pale lavender-coloured bodies. Only a certain proportion of these stained with mucicarmine, but in certain situations, as for example, near the dentate nucleus of the cerebellum and in areas in the occipital white matter, mucinoid masses similar to those in Case I were fairly abundant. With Heidenhain's Azan stain also the picture was slightly different from that in Case I, in that a larger proportion of the masses stained red and a smaller proportion blue. The disappearance of interfascicular oligodendroglia nuclei also was not so complete as in Case I. They were normally present in the spinal cord, and here no mucinoid or lipoid globules were seen. Degenerated nuclei of this kind were also seen in the less affected fibre-systems, for example in parts of the cerebellar hemispheres, in the hilus of the olives,

the reticular formation and transverse fibres of the pons and among the thalamo-striate fibres in the basal ganglia. With Bielschowsky's method a network of fine fibres was seen throughout the white matter of the cerebral hemispheres, in fact there appeared to be little destruction of axis-cylinders. The retinæ were normal.

The changes were thus less pronounced than in Case I and indicated a rather earlier stage in a similar degenerative process. This was brought out especially well with Heidenhain's Azan stain, by which it appeared that the earlier stages of lipoid degeneration were stained red and the later stages blue. Thus in the pons the globules among the pyramidal fibres were chiefly blue and those among the transverse fibres of the pons mostly red. Similarly in Case I the large proportion of red-staining granules in the subcortical zone may be taken as evidence that this zone was last affected.

There was also rather less intense neuroglial overgrowth in this case than in Case I, although in the cerebellum, in spite of the relative preservation of the myelin sheaths, there was a considerable degree of neuroglial sclerosis which affected the molecular layer to an almost equal degree to that seen in Case I. Another interesting observation in the cerebellar cortex in both cases was the paucity of basket-fibres. Otherwise the cell and fibre structure of the cerebellar cortex was well preserved.

DISCUSSION.

These two cases are thus so similar in their clinical and pathological pictures that there can be no doubt that they are two examples of one disease-process. It is equally clear that this process differs definitely and perhaps essentially from that of Schilder's disease. In fact it is only in the rarefaction of myelin sheaths in the centrum ovale and the cerebellar hemispheres and in the compensatory gliosis that the two conditions resemble one another. The condition described approximates closely both in distribution and degree of demyelination to the state of the cerebral hemispheres in a three-months baby. This correspondence is seen not only in the striking contrast of the well-myelinated optic radiations to the unmyelinated areas round them, but also in the state of myelination of the basal ganglia and the pes pedunculi. It is noteworthy also that the lack of myelin in the hemispheres is associated with a fairly well preserved network of axis-cylinders; and the comparative thickness of the myelin sheaths of the pyramidal tracts in the medulla as compared with those in the internal capsule suggests a process of local malnutrition of myelin.

The other striking features about this condition are:—

(1) The diffuse nature of the gliosis, which was noticeable in regions where the myelination was intact although less pronounced here than in the white centres of the cerebrum and cerebellum.

(2) The complete or almost complete absence of nuclei of the interfascicular glia, not only from the demyelinated tracts but also from some of those in which the myelination was still fairly normal.

(3) The presence in enormous numbers of balls of mucinoid and abnormal lipoid material.

In association with these features there was a remarkably slight cellular reaction in the demyelinated areas so that fat-granule cells were only to be seen in the Virchow-Robin spaces. This suggests that no very active breaking down of preexisting myelin was taking place.

The chief problem in these cases is to explain the distribution of the demyelination in relation to the absence of interfascicular glial cells. These cells had obviously degenerated first and the demyelination of fibre-systems appeared to be secondary to this degeneration. Along with their degeneration there was a deposition first of lipoid and later of mucinoid material, which eventually found its way into the Virchow-Robin spaces. To some extent this material might have been derived from the degeneration of the interfascicular oligodendrocytes, although typical ' mucocytes ' of Grynfeltt were absent in Case I and very scarce in Case II.

It is specially noteworthy that the demyelination affected only those tracts in which myelination does not occur till after birth, and was most marked in those systems in which myelination is delayed until after the third month. It is well known that the differentiation of interfascicular oligodendroglia goes hand in hand with myelination. Thus at birth there are numerous oligodendroblasts in the unmyelinated systems but very few mature oligodendrocytes. The explanation of these two cases may therefore lie in a failure of differentiation of oligodendroglia cells and in a greater vulnerability to febrile and metabolic disturbances of these undifferentiated cells. On the other hand the abundant masses of abnormal lipoid and mucinoid material recall the suggestion of Bielschowsky and Henneberg, who also found masses of abnormal lipoid in their cases, that the material from which the myelin is formed might be supplied in abnormal form. The thinness and pallor of the myelin sheaths in the cerebral hemispheres and the absence of any reactive fat phagocytosis, certainly suggested rather a failure of nutrition of myelin sheaths than an active destruction of myelin. It is possible that during fœtal life the nutritive material was normally supplied from the maternal blood, so that up till birth myelination proceeded along normal lines; and after birth this substance may have been to a limited extent available from the mother's milk, so that symptoms did not show themselves until this supply was withdrawn.

In spite of great differences there are certain affinities between these cases and the familial cases of Schilder's disease. In the cases of the latter kind that I have had the opportunity of examining it was noticeable that one of the earliest alterations in the affected areas was a loss of anisotropic

power by the myelin, and that anisotropic granules were very scarce in the fat phagocytes. In the two cases here described there was a similar lack of anisotropic properties. The thickest myelin sheaths were indeed doubly refractile in a faint manner, but no brightly anisotropic material was seen anywhere in the affected areas.

It was noted by Collier and Greenfield that the disease-process in their familial case ran along fibre-systems such as the pyramidal tracts and the anterior commissure. Among the fibres of these systems the abnormal cells called by them ' globoid cells ' were abundant, although no such cells could be seen in neighbouring unaffected areas.* In the two cases here described there was also an affection of fibre-systems, although the histological picture in the affected systems bore no resemblance to that described by Collier and Greenfield. Here one must again insist on the fact that mucinoid globules were found among the pyramidal tract fibres throughout the brain-stem, but not (in Case II at any rate) in the spinal cord. This fact at once distinguishes the degeneration of the pyramidal tract in these cases from Wallerian degeneration due to a lesion in the white matter of the hemispheres.

Whether these cases were familial is not at present definitely known. The history of the eldest child in the family of Case II was so similar to that of our patient that it appears probable that he was suffering from the same condition. But it is possible that the diagnosis of hydrocephalus in his case was the correct one. As Case I was a first born and only child no evidence of familial incidence is here available. In any case the condition appears to be of a purely degenerative character and to be in some degree at least a developmental error revealing itself in the early years of life.

SUMMARY.

1. Two cases of cerebral sclerosis occurring in young children are described. Both patients died at the age of three years; in one case there was some indication that a previous member of the family had been similarly affected.

2. The demyelination in these cases affected the white matter of the cerebral hemisphere diffusely, but the optic radiations and the pyramidal tracts were relatively spared. The fibres which myelinate latest appeared to be primarily affected.

3. The interfascicular oligodendrocytes were absent in the case with the longer history, and very degenerated in that with the shorter history. Along with this there was a remarkable accumulation of masses of mucinoid

* N.B.—These cells differed in many respects from the normal reacting types of neuroglial cell (gemästete Gliazell) although we considered that they were of neuroglial origin. Prof. Spielmeyer, however, who kindly examined our sections, thought that they were of mesodermal origin, and Carnegie Dickson, who has found similar cells in the younger members of the same family, is of a similar opinion.

and abnormal lipoid granules. These were found throughout the pyramidal tract in the brainstem and in other partially demyelinated fibre-systems as well as in the most demyelinated areas.

4. The pathogenesis of the condition appears to be failure of nutrition of the myelin sheaths owing to the degeneration of the interfascicular glia. The latter may be due to faulty development of those oligodendrocytes which are differentiated last.

5. It is suggested that the primary cause of the condition may be the child's inability to form the proper lipoid material for the formation of myelin.

It is a pleasure to thank Drs. Collier and Critchley for their permission to publish these cases and Professor Lucas Keene and Dr. Evelyn Hewer for valuable assistance on the myelination of the developing human brain.

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SIZE IN MENTAL DEFICIENCY.*

By

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The present study has been undertaken with two objects in view: first, to ascertain how size varies among fully grown defectives at different mental levels, and secondly, to provide a satisfactory background for further studies of particular parts, and especially of the nervous system.

In the past, various workers, searching for a neural basis of amentia, have measured, say, the thickness of the cerebral cortex or the brain volume, and, finding a diminution, have at once assumed that this must be causally related to the deficiency in intelligence. But clinical experience tells us that defectives are nearly always undersized in their entire somatic development, so that the diminution of any one part may be merely a local expression of a perfectly general shrinkage. This, of course, will not affect the data as such, but it may modify our deductions from those data. Clearly, if we are to be certain that the falling off in size of one particular part is not merely part of a general change we must first of all know what is happening elsewhere. To put it briefly, it is best, before studying particular parts, to study the whole. The present work is a general survey of size in patients who exhibit different degrees of intelligence ranging from complete idiocy up to the border line which separates the feebleminded from the subnormal.

THE MATERIAL.

The material consists of 269 male defectives, their ages being in all cases above sixteen years. Senile aments and those with paralysis or definite malformations of the limbs and trunk were excluded. Hydrocephalics were not included, since in this condition head measurements must be regarded just as much an index of water volume as of brain size. On the other hand, microcephalics, whose small brains are but seldom the seat of ventricular dilatation, have been included. Whether or not the microcephalic belongs to a definite clinical type is a question with which we are not here concerned.

PART I.

LINEAR HEAD MEASUREMENTS IN MENTAL DEFICIENCY.

Two hundred and sixty-nine defectives of varying mental age were measured for head length, head breadth and ear to ear measurements.

Head length was measured by calipers from the most prominent point of the glabella to the occipital point. The measurement was made with Flowers type calipers and was read to the nearest quarter centimetre. Head length was measured with calipers to the nearest quarter centimetre at the

* Towards the expenses of this research a grant was made by the British Medical Association.

widest part of the head above the plane of the ear-holes. 'Ear to ear' was measured with a tape-measure from the centre of each external auditory meatus, vertically over the top of the head. It was read to the nearest eighth-inch.

Head height proved in practice to be difficult to measure with reasonable accuracy. It was considered equally accurate to use a combination of head breadth and 'ear to ear,' assuming the coronal section of the head be half an ellipse. This gives the equation:—

$$\text{Head height} = \frac{2}{\pi}(\text{ear to ear}) - \frac{\text{head breadth}}{2}$$

Using this equation, head height was calculated to the nearest quarter centimetre.

Tables I–IV (see appendix) give the distributions of these measurements at different mental ages.

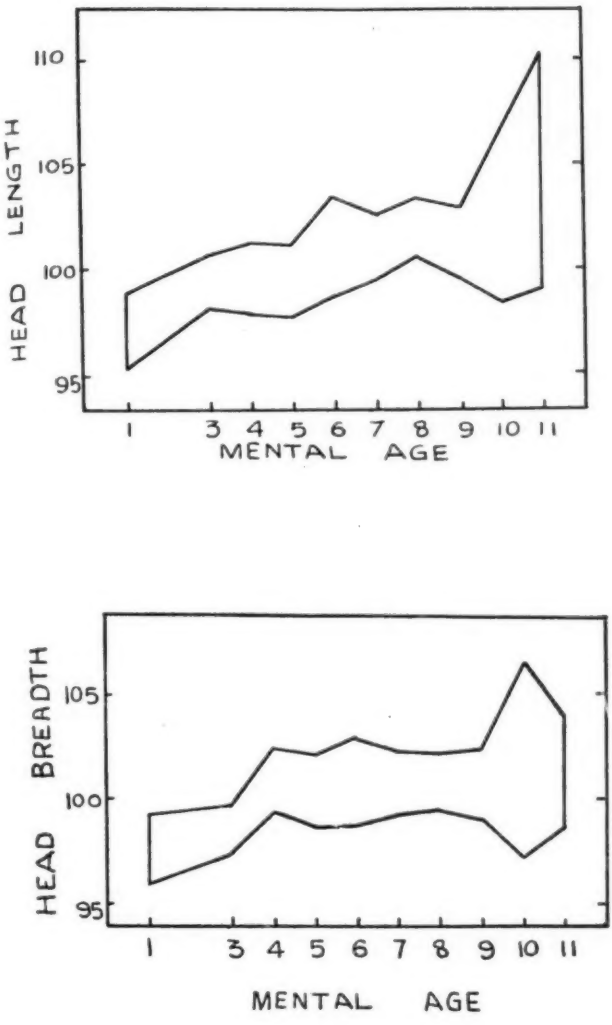
We may now proceed to extract the information in the following way. So as to be able to compare one measurement with another, say head length with head breadth, we first convert the data to bring all the general means to 100. This gives us the data in a form where the relative changes due to intelligence are left unaltered, but at the same time are made comparable between different measurements. We then find the average for each measurement at each mental age.

Now these means, say mean head length at mental age 4, are exact for that particular group of patients of mental age 4 which we have measured. But this group is only a sample of what we would have obtained had we measured all possible people of mental age 4. Another sample would give a slightly different result. It is not possible to say what the true mean really is, but it *is* possible to say within what limits it must lie. In fact, in work of this type, it is often much more important to know the limits within which it must lie than to know merely what our particular sample has as mean. Thus in head length, mental age 1 group, we can be sure that the true mean lies between 95·3 and 98·8, and this is much more informatory than merely to know that our mean happens to be 97·051.

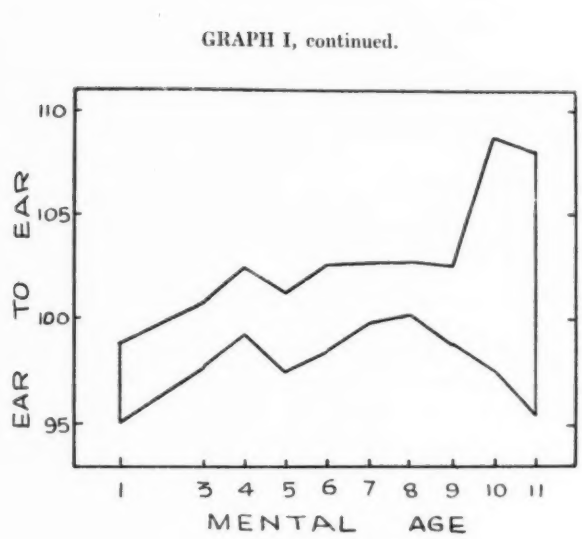
We have therefore expressed the data in such a way as to present the limits within which the true mean must lie.

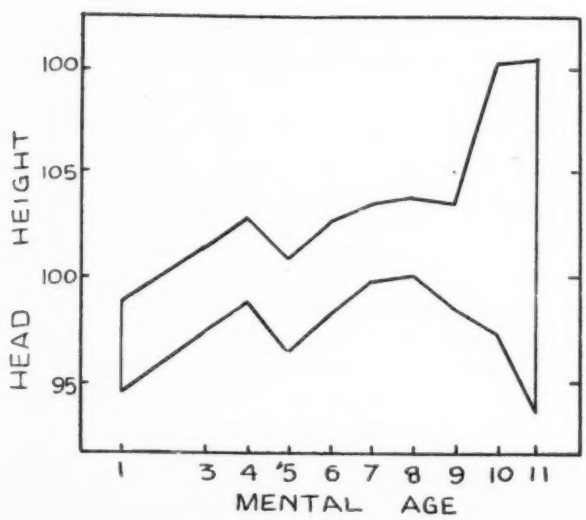
The 'ribbon' representation in graph I shows the change in size at different mental ages for the four measurements. It shows a strip within which we can be sure that the true line of means will run. It is much preferable to the usual method of plotting a more or less irregular line, for the latter gives no clue as to what deviations from the line are statistically permissible. It will be seen in the graph, for instance, that the right-hand ends are trumpet-shaped. This is due to the fact that as the number of cases becomes fewer at this end, the mean is not specified with so much precision, and this is allowed for exactly by the opening out of the limiting lines.

GRAPH I.


HEAD LENGTH
110
105
100
95
1 3 4 5 6 7 8 9 10 11
MENTAL AGE
HEAD BREADTH
105
100
95
1 3 4 5 6 7 8 9 10 11
MENTAL AGE

GRAPH I, continued.





From the graph we may note, firstly, that mental age certainly has some effect on head size since we cannot draw a horizontal line to run through the ribbon. It is clear that the left-hand end must turn down. As intelligence falls off (to the left), we see that head size, as shown generally in all the measurements, falls off also, but the falling off in size is most marked at the lower (idiot) end. It is important to note that the general form of the curves is similar.

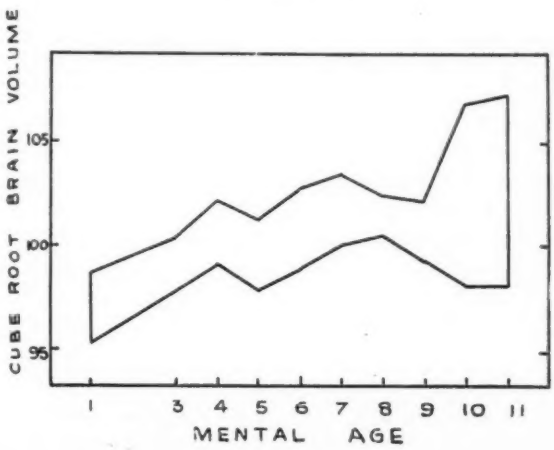
In addition, an index of brain volume was calculated by multiplying together head length, head breadth and head height and dividing by 1,000 for convenience. There is no point, in this investigation, in applying one of the formulæ, such as Lee's, for finding the most probable brain volume in cubic centimetres. Since we wish merely to compare group with group, an index will serve the same purpose equally well.

The actual distribution of the brain volume index is given in the appendix (table V). Treating the data as before, we find the graph to be similar to those above but to have a much steeper slope. This, however, is to be expected since here we are dealing with volume (three-dimensional), while previously we were dealing with linear (one-dimensional) quantities. In this case we would expect the volume measurement to have a slope about three times as steep as the others.

To make the brain volume measurements comparable with the others, we must either cube the latter, or take the cube root of the former. Since linear quantities are the easier to handle, we will take the cube root of the brain volume as the linear representative of the data (appendix, table VI).

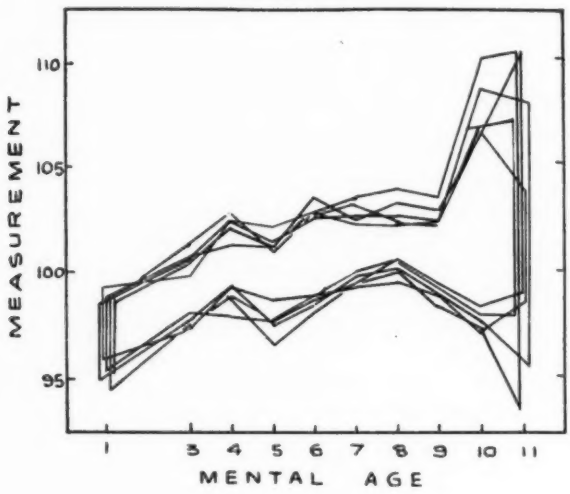
Treating the new data as before, we find the following (graph II).

GRAPH II.



This, it will be seen, has much the same slope and general form as those of the other head measurements. All five curves seem to be similar, and if plotted on top of one another we obtain the following (graph III).

GRAPH III.



The curves are all obviously similar, except for the small random sampling irregularities which are to be expected.

CONCLUSIONS.

With regard to head measurements generally, we may note that the following points are established by the data:—

(1) Head size falls off generally with decreasing intelligence.

(2) The falling off in size is quite general and affects equally head length, head breadth, head height, and ear to ear measurements. Brain volume, when made comparable, also alters in just the same way.

(3) The falling off is most marked in the ' idiot ' region.

Further conclusions must be postponed till after Part II has been considered.

APPENDIX TO PART I.—TABLES OF DATA AND STATISTICAL METHODS USED.

THE MEASUREMENTS.—*The general effect of errors in measurements.*

In work of this type, there are three types of errors which may be present. They are (1) biased, (2) random, (3) correlated. Their effects on the data will be quite different.

(1) Biased errors, i.e. reading all measurements too high or too low, will have no particular effect, since we want simply to compare group with group. Such comparison is unaffected by a systematic change.

(2) Random errors refer to the small discrepancies between repeated measurements which are always found in any series of measurements. Associated with them are the errors introduced by grouping, i.e. by measuring 'to the nearest unit.' Random errors introduce a certain extra 'blurriness' to the data. It must be remembered, however, that in biological data there is always a certain blurriness of the data in any case. The 'scatter' is measured by the variance. Random errors have the effect of increasing the variance. If we are comparing group with group so as to compare the means, then the effect of introducing random errors is precisely the same as if we had dealt with a smaller number in our sample—that is to say, it is equivalent to wasting some of our cases. In the present series, the random and grouping errors waste about one per cent. of the total information—that is to say, our series of 269 cases measured with our technique and grouping is equivalent to a series of 266 cases measured with infinite accuracy. It does not appear to be generally realized that the effect of increasing accuracy can be greatly overestimated in some types of biological work.

In all the measurements, the results have been grouped to a unit equal to about a third or a quarter of the standard deviation. This means that, in grouping units, the variance is about 9 to 16 (squared) units. The variance introduced by grouping is 0·083. The latter is less than one per cent. of 9, so that the wastage of information is less than one per cent., as stated above.

The discussion of errors above refers to errors of measurement of size. In the case of the measurement of intelligence there is also bound to be some error. Most cases were measured with Burt's Revision of the Binet-Simon test, some with Porteus' maze, and some with both. In the latter case the two estimates were averaged. The Porteus' maze seems to give results a little higher than Burt's Revision, but this error cannot affect the systematic differences between the mental-age groups, since the maze tests were scattered more or less at random over all mental ages.

The general effect of small errors in measuring mental age will be to diminish the differences between the measurements obtained at the different age groups. But since the variance in measuring mental age would be fairly constant in different investigations, while on the one hand it has the effect of lowering the differences found between various mental ages, yet these differences will still be comparable with one another, both between different investigations and between different investigators.

Owing to the fact that there are no suitable tests for very low-grade patients, the cases with mental age of less than three years (0, 1 and 2 years) have been grouped.

The measurements have all been done by one person.

It may be objected that in this series there are no normal controls. These have been omitted deliberately for two reasons. Firstly, the series does not need outside controls, since we are comparing the mental age groups with each other, and each group acts as control to the other. The normals, if used at all, would be used not so much as controls, but rather to extend the range of mental age. They have not been included here owing to the practical impossibility of obtaining comparable material. To use nurses, for instance, would be obviously absurd, since they are deliberately selected for size. It was considered safer to leave the range restricted than to attempt to extend it by including material of doubful validity. To include a group which is not comparable would be to vitiate the whole work.

There is only one other possible source of error. It is that this institution, in admitting patients, may tend to take certain types, such as small and feeble idiots, or large and strong feeble-minded patients. In such a case, our collection of idiots, for instance, would tend in size to be below the general average of idiots. From a knowledge of the administrative side, however, we are satisfied that no such selection of types exists with regard to this institution.

TABLE I.

The distribution of *head length* for various mental ages is:—

Head length in cm.	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
21·75	...	...	...	1						
21·50										
21·25	...	...	...	...	1					
21·00	...	...	...	...	1	...	..	...	1	
20·75	...	...	1	1	2					
20·50	...	...	2	...	1	...	2			
20·25	...	3	1	2	1	1	...	2	1	1
20·00	1	1	1	1	1	2	2	...	...	2
19·75	2	2	2	...	3	1	4	2		
19·50	5	3	3	1	3	6	5	3	1	
19·25	1	8	4	3	1	3	...	2	1	
19·00	3	4	7	6	2	4	4	3	2	
18·75	3	5	3	6	2	2	3	1	1	1
18·50	4	8	...	7	1	2	4	4		
18·25	4	3	4	2	4	1	2	1		
18·00	4	1	2	1	2	2	...	1	1	
17·75	1	2	2	2	1					
17·50	1	2	1	2	...	1				
17·25	5	2	4	...	2					
17·00	2	...	1							
16·75	...	...	...	1	1					
16·50	...	...	...	1						
16·25	1	1								
16·00	...									
15·75	1									
	38	45	38	37	29	25	26	19	8	4

Head breadth in cm.

TABLE II.

The distribution of *head breadth* for various mental ages is:—

Head breadth in cm.	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
16·75	...	...	...	1						
16·50										
16·25	...	...	1	...	...	...	...	...	1	
16·00	1	1	2	1	2	1	1	...	1	
15·75	...	1	..	2	4	..	...	1		
15·50	2	2	4	4	2	3	1	1	1	
15·25	2	1	6	2	3	3	5	4		
15·00	6	3	7	3	4	6	4	3	...	3
14·75	..	8	3	9	2	1	5	3	1	
14·50	7	12	6	4	2	3	7	2	2	1
14·25	7	6	5	4	3	7	2	3	1	
14·00	4	5	1	4	4	...	...	1	1	
13·75	4	4	...	1	1	1	...	1		
13·50	...	...	1	...	2	...	1			
13·25	3	1	2	1						
13·00	...	1	...	1						
12·75	2									

TABLE III.

The distribution of '*ear to ear*' for various mental ages is:—

'Ear to ear' in inches	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
15·750	...	...	1							
15·625										
15·500	...	...	...	...	...	...	...	...	1	
15·375	...	1								
15·250	...	...	1	2	1	...	1			
15·125	..	...	...	...	1	...	...	...	1	
15·000	1	1	...	...	1	...	...	..	1	
14·875	...	...	1	1	1	...	...	1	...	1
14·750	...	...	2	...	1	1				
14·625	2	2	2	1	1	3	2	2		
14·500	...	3	2	2	1	...	1			
14·375	1	2	1	3	...	3	3	1		
14·250	2	4	3	2	3	4	2	2	1	
14·125	3	3	5	1	3	2	4	4	1	
14·000	4	2	...	3	2	2	4			
13·875	2	3	2	3	2	1	1	2	1	1
13·750	1	3	5	5	...	3	2	1	...	2
13·625	1	2	3	...	2	1	2	1		
13·500	...	...	2	3	1	2	1	...	1	
13·375	...	2	1	1	1	2	1	1		
13·250	1	7	4	2	3	...	1	2		
13·125	6	1	...	2	2	...	1	1		
13·000	4	2	1	1	1	...	...	1		
12·875	2	3	...	...	1					
12·750	1	2	1	1	...	...	...	...	1	
12·625	2	1	...	...	...	1				
12·500	1									
12·375	...	...	...	1						
12·250	1	...	1	2						
12·125	1	1								
12·000	1	...	...	...	1					
11·875	...	...	...	1						
11·750										
11·625										
11·500	1									

Head height in cm.

TABLE IV.

The distribution of *head height* for various mental ages is:—

Head height in cm.	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
17·50	...	1	1							
17·25										
17·00	...	...	...	...	...	...	1	...	1	
16·75	...	1	1	1	1	...	...	1	1	
16·50	...	...	1	...	2	...	...	...	...	1
16·25	2	3	1	3	...	1	1	1	1	
16·00	1	1	6	1	2	5	3	2		
15·75	2	6	2	2	2	2	4	1	1	
15·50	6	6	1	3	4	6	3	4	1	
15·25	2	3	7	6	4	2	3	1	1	
15·00	3	2	3	2	2	...	5	1	...	2
14·75	2	2	3	5	3	5	3	2	...	1
14·50	...	4	4	3	1	1	1	2	1	
14·25	4	7	5	4	5	2	...	2		
14·00	6	2	...	1	2	...	1	2		
13·75	1	2	1	1	...	...	1			
13·50	2	3	.	1	...	1	...	...	1	
13·25	4	1	2	1						
13·00	1	1	..	1						
12·75	1	...	..	2	1					
12·50	1									

TABLE V.

The distribution of the *brain volume* index for various mental ages is:—

Brain volume index	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
5·7	...	...	1	2						
5·6										
5·5	...	...	...	...	2	...	...	...	1	
5·4	...	..	1							
5·3	...	...	1	...	1	...	1			
5·2	...	...	...	...	1	...	...	...	2	
5·1	1	...	...	1	1					
5·0	...	...	1	...	...	1	...	...	...	1
4·9	...	3	1	...	1	1				
4·8	1	...	2	1	...	2	2	1		
4·7	2	2	4	2	2	2	1	2		
4·6	...	3	...	1	3	1	3	2		
4·5	3	5	2	3	1	...	4	3	...	1
4·4	2	3	3	2	1	3	2	...	...	1
4·3	1	3	3	1	2	4	1	4	2	
4·2	1	2	1	1	2	1	4	1	1	
4·1	3	3	4	9	3	4	4	1	...	1
4·0	2	3	2	2	...	1	1			
3·9	...	1	1	2	...	2	3	1		
3·8	2	3	2	2	3	...	...	2	1	
3·7	4	3	4	1	1	1	...	1		
3·6	4	2	2	1	2	...	...	1	1	
3·5	3	3	1	...	1					
3·4	2	3	1	...	...	1				
3·3	1	1	...	2						
3·2	2	...	...	2	1					
3·1	1	2	...	1						
3·0	...	...	1	...	...	1				
2·9	1	...	...	1	1					
2·8	1									
2·7										
2·6										
2·5										
2·4	1									

TABLE VI.

The distribution of *cube root-brain volume* for various mental ages is:—

Cube root of brain volume	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
1·80	...	...	0·2	0·4						
1·78	...	...	0·8	1·6						
1·76	...	...	1·0	...	2·0	...	...	..	1·0	
1·74	..	...	1·0	...	1·7	0·5	1·0	...	1·4	
1·72	1·0	...	0·5	1·0	1·3	1·9	...	...	1·6	0·5
1·70	0·2	3·0	1·9	0·2	1·0	3·6	0·4	0·2	...	0·5
1·68	2·8	2·0	5·6	2·8	2·0	1·0	2·6	2·8		
1·66	1·8	6·0	1·2	2·8	3·6	3·8	5·4	3·8	...	0·6
1·64	3·4	5·6	4·4	3·4	1·8	4·0	3·8	2·0	0·4	1·4
1·62	1·6	4·0	3·2	1·6	3·2	4·5	4·0	4·0	2·4	
1·60	3·8	4·3	4·8	9·8	3·4	2 3	5·1	1·2	0·2	1·0
1·58	1·4	2·9	2 2	3·0	...	0·4	3·1	0·8		
1·56	3·2	4·1	3·4	2·7	3·3	0·3	0·6	2·5	1·0	
1·54	5·6	3·5	4·2	1·4	2·1	0·7	...	1·4	0·7	
1·52	4·2	3·6	1·6	0·3	1·6	...	...	0·3	0·3	
1·50	2·4	3·4	1·0	0·8	...	1·0				
1·48	2·0	0·6	...	2·6	0·7					
1·46	1·6	2·0	...	1·6	0·3					
1·44	0·3	...	1·0	0·3	0·3	1·0				
1·42	1·1	...	...	0·7	0·7					
1·40	0·6									
1·38										
1·36										
1·34	1·0									

In the table above, the distribution gives what would have been obtained had the root been obtained from the original (ungrouped) data. This gives fractional frequencies, but avoids the systematic error which would be introduced by taking the root from the data after grouping.

We now proceed to reduce the figures to a common basis. This may be done in several ways. For our purpose, probably the best way is to multiply the measurements by some factor so that the general means all come to 100. Thus, the general mean of all head length measurements is 18·873 cm. Consequently if we multiply all the measurements by 100/18·873 the general mean will come to 100. This method makes a more effective use of the data than, say, trying to adjust them to some rather hypothetical normal.

We find the means and variances in each column directly from the tables of distributions and correct them to the standard form by multiplying them

by 100/18·873 and $(100/18{\cdot}873)^2$ respectively. Thus for head length, mental age 1 year, we find the standardized mean to be 97·05 and the corresponding variance to be 28·95.

To compare these means, it is probably best to keep all the mental age groups quite separate. One could fit a polynomial and compare these to find the trend, but to apply any test of significance we would have to assume that the residual variance was constant. It appears from the data that this is definitely not so.

If we were going to use some further analysis of the data, then we would have to use a polynomial fitted by least squares, otherwise any tests of significance applied would be inaccurate. In our case, however, we merely need the form of the regression line, and this can be better obtained by a free hand curve. If we were to fit a polynomial, we would have the further disadvantage that a parabola would be too arbitrary, while a cubic or curve of higher degree would show too much tendency to waggle.

The method adopted here is to keep each mental age group quite separate and to compare group with group and measurement with measurement simply by direct comparison, using the standard deviation as a test of significance. This method is not so sensitive as a method using compounded data, but the latter may in our case be too sensitive and might give erroneous conclusions.

To find the limits of the mean we calculate the standard deviation of the mean. Then, using ' Student's ' t distribution, we find the distance of the 5% point from the mean. Thus, for mental age 1, $n' = 38$, $n = 37$, 5% point = 2·02. Multiplying the standard deviation of the mean by this, we then add and subtract this from the mean and thus find the (5%) limits within which the true mean is almost certain to lie.

We thus obtain the following limits for the means:—

TABLE VII.

Mental age in years	Head length	Head breadth	Ear to ear
1	95·3— 98·8	96·0— 99·3	95·0— 98·8
3	98·1—100·8	97·4— 99·8	97·7—100·7
4	97·9—101·3	99·4—102·5	99·3—102·6
5	97·7—101·2	98·7—102·1	97·5—101·3
6	98·7—103·5	98·9—102·9	98·5—102·7
7	99·6—102·6	99·3—102·3	99·9—102·8
8	100·6—103·4	99·5—102·2	100·2—103·0
9	99·6—102·9	99·0—102·4	98·9—102·7
10	98·4—106·6	97·2—106·7	97·7—108·8
11	99·0—110·3	98·7—103·9	95·5—108·0

Mental age in years	Head height	Brain volume	$^3\sqrt{}$ Brain volume
1	94·4— 98·9	87·0— 96·4	95·3— 98·7
3	97·6—101·4	93·9—101·0	97·8—100·3
4	98·9—102·9	97·4—106·6	99·1—102·1
5	96·6—100·9	93·9—103·9	97·8—101·2
6	98·2—102·7	97·1—109·1	98·9—102·8
7	99·8—103·6	97·4—106·6	100·0—103·4
8	100·1—103·9	101·3—107·7	100·5—102·5
9	98·5—103·6	98·2—106·6	99·4—102·2
10	97·3—110·2	94·1—122·1	98·1—106·9
11	93·6—110·5	93·6—122·0	98·1—107·2

PART II.

BODY WEIGHT, ARM LENGTH AND FOOT LENGTH IN MENTAL DEFECTIVES AT DIFFERENT MENTAL AGES.

Continuing the investigation of size in mental deficiency, we have also measured body weight, arm length and foot length, so as to obtain an idea of what the rest of the body, apart from the head, is doing.

THE METHODS.—In examining the weight record of each patient, it was obvious that each person's weight is rather variable, there being steady upward and downward trends for no particular reason. 'Body weight' was therefore obtained by taking a fair average value of the monthly weight record of each patient. The weights were afterwards grouped to the nearest 7 lbs.

The right arm and right foot were measured in 256 of the total series, the original number being reduced owing to transfers or the unsuitability of the subjects.

The arm was measured from the olecranon to the tip of the styloid process of the ulna. These limits were chosen as they can be identified with certainty and terminate in comparatively sharp points.

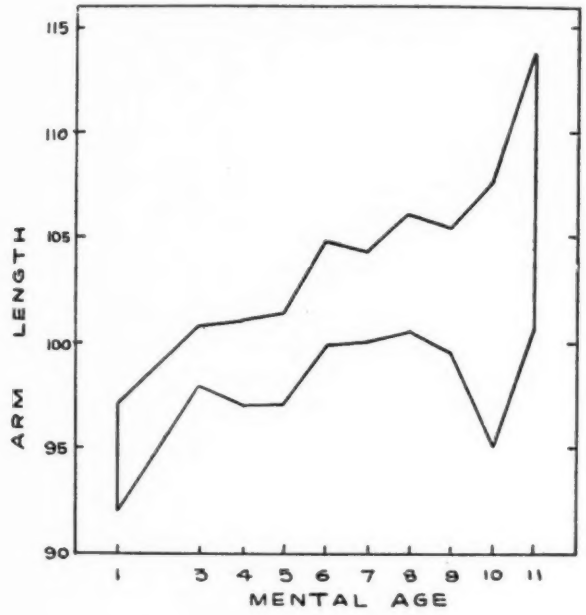
The foot was measured from the heel to the tip of the big toe. The instrument used resembled that employed by the shoemaker for measuring feet, but the scale was graduated specially to read to the nearest quarter centimetre. This grouping, however, proved unnecessarily fine and the measurements were subsequently regrouped.

The general discussion of the question of errors was given in Part I and need not be repeated. Exactly the same principles are applicable here.

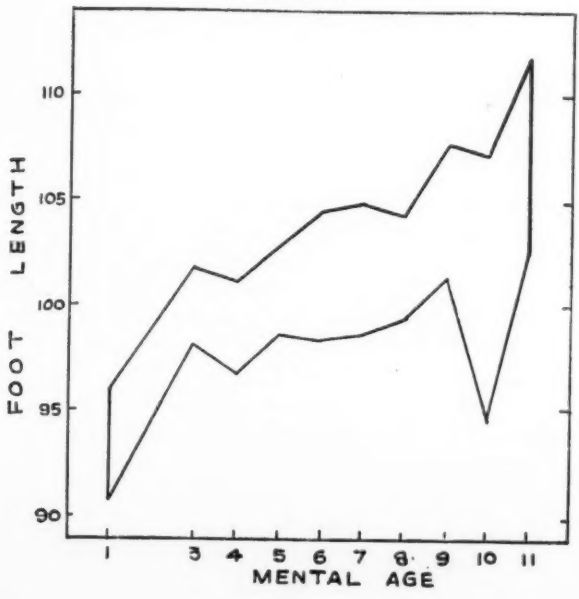
The full data and statistical methods used are given in the appendix as they are not of general interest.

Analyzing the data as before (Part I), and graphing the results, we find the mean lengths at various mental ages to be, for arm and foot:—

GRAPH 1.



GRAPH 2.

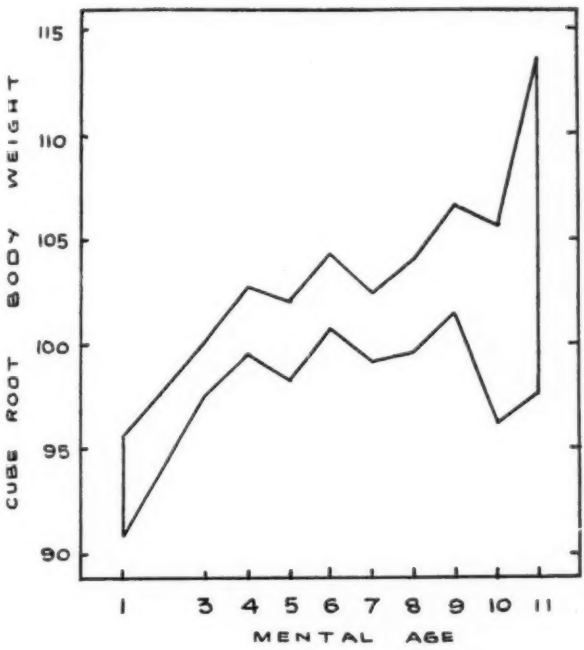


To obtain the graphs, we have taken the average at each mental age, and have plotted, not the actual averages found in our sample, but the limits within which the true mean must lie. In this way one is able to assess the significance of changes much more easily than if one were given merely the line of the actual averages. The ' ribbon ' means that the true line must run somewhere inside it.

The ' body weight ' results are similar but are much steeper in slope. This is to be expected since weight is a ' cubic ' unit, while ' length ' is linear. One would expect ' weight ' to have about three times the slope of ' length.' To make the results comparable to the arm and foot results, we may take the cube root of body weight and use this as our estimate of size.

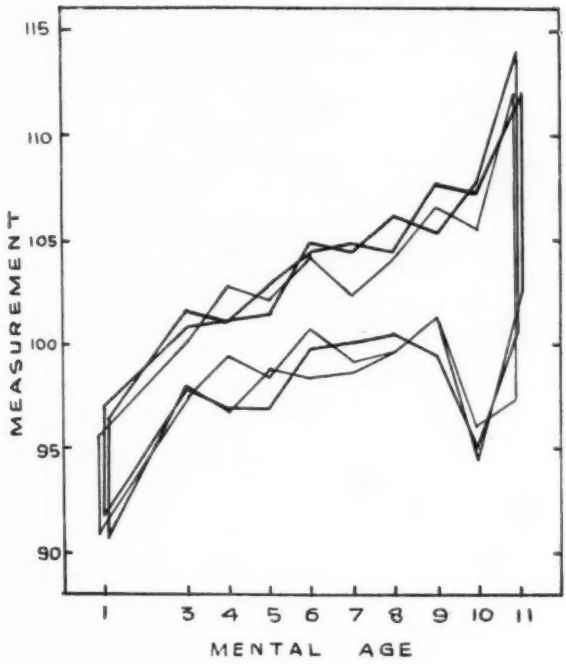
Dealing, not with body weight, but with its cube root, we find this quantity to vary with intelligence as shown in the following graph:—

GRAPH 3.



It will be seen that the general form of the three curves is similar. This is confirmed by plotting all three curves on top of each other.

GRAPH 4.



The three curves are entirely similar except for small non-systematic, random sampling irregularities.

From the graph above, we may draw the following preliminary conclusions:—

(1) All three body measurements change with alterations of mental age. Measurements of patients of lower mental age tend to be smaller than those with higher mental age.

(2) All three measurements seem to change equally, i.e. the shrinkage appears to be perfectly general and not limited to any particular part of the body.

(3) The falling off in size is most marked in the ' idiot ' region, but there is clearly (graph 4) some falling off in the higher grades.

These conclusions are very similar to those obtained from an examination of head measurements (Part I). We will therefore compare the four head with the three body measurements.

On examining the data (see appendix B), it is found that the data of Parts I and II fall into two distinct groups:—

(A) Head length, head breadth, ' ear to ear ' and cube root brain volume.

(B) Cube root body weight, arm length and foot length.

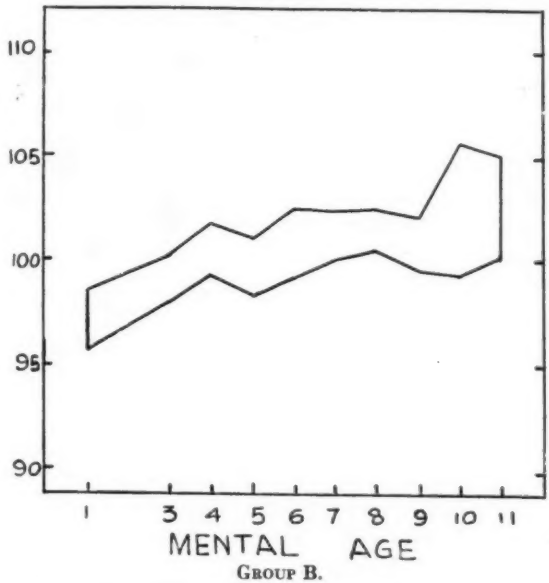
In group A, the different measurements all seem to vary with mental age in the same way, and the three measurements in group B are also similar to

one another, but group A shows differences from group B. The latter has generally a steeper slope than group A. This may be shown by combining the four measurements in group A together and the three in group B (see appendix C).

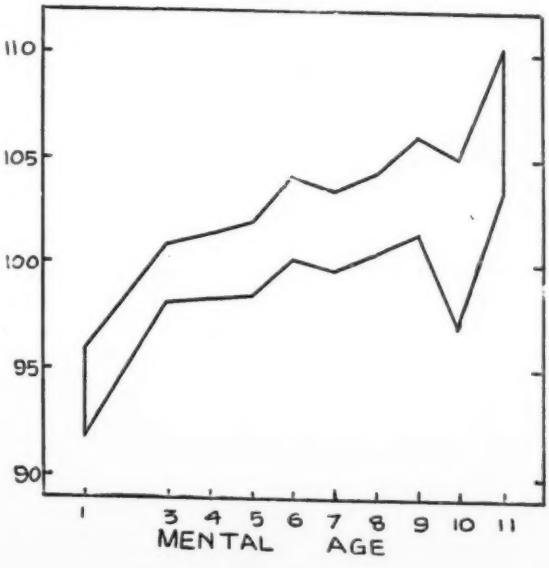
Group A and group B are graphed below:—

GRAPH 5.

GROUP A.



GROUP B.



A shows what happens when all the head measurements are combined together. B shows the combined body measurements.

It will be noticed that this grouping, which has been found simply by an examination of the numerical data, also corresponds to a natural anatomical grouping, since group A consists of all the measurements related to the skull or brain, while group B contains the measurements obtained from the rest of the body.

An examination of the above graph shows that the ' body ' group of measurements (B) actually changes more for a given change of mental age than does the ' brain ' group of measurements (A). This difference in slope is statistically significant (appendix B).

Before we sum up, we may first state that throughout the paper, before discussing any finding, we have first made sure, by actual testing, that the finding in question is statistically significant. The reader may rest assured that we are not discussing any points which are really insignificant.

To sum up Parts I and II, we see that consideration of the above data establishes the following points:—

(1) All the measurements made change with mental age, all decreasing with lower intelligence.

(2) When the measurements are reduced to a comparable basis, the forms of the curves are roughly the same, all showing at first a slight fall, and then a more rapid fall as idiocy is reached.

(3) The measurements fall into two natural groups which correspond with the division into ' head size ' indices and ' body size ' indices. These two groups show significant differences between them.

(4) As intelligence falls, body size falls off more rapidly than head size.

GENERAL CONCLUSIONS.

(1) When we come to consider the general significance of these findings, a point of importance at once emerges. If there is present a general change of size with intelligence, then it follows that attempts to localize intelligence by the employment of methods which restrict the investigation to a comparison of measurements made on defectives with the same measurements made on normal subjects can have only a limited value. Further, the data they provide must be interpreted with the greatest caution. To measure some part—perhaps the cerebral cortex—and having found that it increases as intelligence increases, to conclude that the part concerned must therefore be connected causally with intelligence is clearly an assumption which cannot be upheld. To use a reductio ad absurdum, we have found that foot length falls off with decreasing mental age, yet no one would accept the conclusion that intelligence must be localized in the feet! Further, we have seen that ' body ' measurements actually fall off more than ' brain ' measurements, but this does not indicate that intelligence is more a property of the body than of the brain.

For the method commonly in vogue which we have referred to above, we must therefore substitute one which will yield more precise information. Although in this paper we are not primarily concerned with technique, yet it may be mentioned that the method of partial correlations provides a better approach to the statistical treatment of the subject.

(2) The data show, as clinical experiences teaches, that the deficiency manifests itself just as much in the body as in the brain, if not more. Mental deficiency, therefore must be regarded as essentially a general phenomenon, affecting the whole individual, rather than a localized disturbance affecting merely one small part.

(3) The fact that as we go down the intelligence scale the body shrinks faster than the brain, is compatible with the hypothesis that mental deficiency is essentially an arrest of development. We may recall that as the various stages of growth are retraced in the normal from childhood to infancy so we also find that the body shrinks faster than the brain.

APPENDIX TO PART II.—TABLES OF DATA AND STATISTICAL METHODS USED.

APPENDIX A.

TABLE 1.

The distribution of *body weight* for various mental ages is:—

Body weight in lbs.	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
175	...	...	...	...	...	...	...	2		
168										
161	...	...	1	1	...	...	...	1	...	1
154	...	...	1	1	2	1	3	1		
147	...	1	4	1	2	1	1	...	...	1
140	...	1	2	5	5	...	3	3	2	
133	1	3	3	2	4	4	5	2	2	
126	4	9	4	6	3	5	3	3	1	1
119	3	6	8	4	6	3	2	2	...	1
112	6	9	8	4	4	4	2	3		
105	3	5	3	7	1	6	4	2	1	
98	4	6	2	3	1	1	2	...	1	
91	1	3	1	2	1	...	...	...	1	
84	7	2	1							
77	3	...	...	...	...	...	1			
70	3									
63	2	...	...	1						
56										
49	1									

TABLE 2.

The distribution of *cube root-body weight* for various mental ages is:—

Cube root body weight	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
5·6	...	...	...	...	...	...	...	2·0		
5·5	...	...	0.5	0.5	..	...	...	0·5	...	0·5
5·4	...	...	1.2	1·2	1·4	0·7	2·1	1·2	...	0·5
5·3	...	0·8	3.5	1.1	2·2	1·1	1·7	0·3	...	0·8
5·2	...	1·2	2.8	5.2	5·4	0·2	3.2	3·0	2·0	0·2
5·1	1·8	4·8	3.8	3.2	4·6	5·0	5·6	2·6	2·2	0·2
5·0	3·8	8·4	4.8	5.6	3·6	4·6	2·8	2·8	0·8	1·0
4·9	4·2	7·5	8.8	4.4	6·0	3·6	2·2	2·5	...	0·8
4·8	4·8	7·3	6.2	4.2	3·0	4·0	2·2	2·5	0·2	
4·7	3·2	5·2	2.8	6.2	1·0	5·0	3·6	1·6	1·0	
4·6	3·2	4·8	1.6	2.4	0·8	0·8	1·6	...	0·8	
4·5	1·0	3·0	1.0	2.0	1·0	..	...	...	1·0	
4·4	4·9	1.4	0.7							
4·3	3·6	0.6	0.3	...	...	...	0·5			
4·2	2·4	...	...	...	...	...	0·5			
4·1	2·1									
4·0	1·4	...	...	0.7						
3·9	0·6	...	...	0.3						
3·8										
3·7	0·5									
3·6	0·5									

TABLE 8.

The distribution of *arm length* for various mental ages is:—

Arm length in cm.	Mental age in years									
	1	3	4	5	6	7	8	9	10	11
30·375	...	...	...	1						
29·875	...	...	...							
29·375				1						
28·875	...	...	...	...	1	...	1	1	...	1
28·375	1	...	1	...	...	1	3	...	1	
27·875	...	1	1	1	6	2	1	3		
27·375	...	1	2	3	3	4	4	1	2	1
26·875	4	2	3	...	2	2	2	...	...	1
26·375	...	10	2	2	2	3	4	4	1	1
25·875	4	6	2	5	4	5	1	4	1	
25·375	4	8	5	6	1	1	...	2	1	
24·875	2	6	8	2	5	4	2			
24·375	3	3	1	8	1	...	2	1		
23·875	4	1	5	2	1	2	2	1		
23·375	2	4	2	3	...	1	1	...	1	
22·875	4	...	...	...	1	...	...	1	1	
22·375	2	2	...	2	1					
21·875	3									
21·375	2	...	1							
20·875	2									

TABLE 4.

The distribution of *foot length* for various mental ages is:—

		Mental age in years									
		1	3	4	5	6	7	8	9	10	11
Foot length in cm.	27·875	...	...	...	...	...	...	...	2		
	27·375	...	...	...	2						
	26·875	...	...	1	...	2	1	1			
	26·375	...	1	1	1	1	3	...	1	...	1
	25·875	...	2	...	...	3	2	3	...	3	
	25·375	...	5	3	3	2	1	...	2	...	1
	24·875	4	5	3	2	4	3	2	5	...	2
	24·375	2	3	2	6	4	3	5	2		
	23·875	4	9	2	8	1	2	4	2		
	23·375	1	3	4	1	3	3	5	1	2	
	22·875	3	6	6	6	...	2	1	2	1	
	22·375	4	3	3	3	2	2	1	1	1	
	21·875	4	1	7	2	2					
	21·375	4	3	...	2	1	...	...	..	1	
	20·875	4	2	1	...	2	3	1			
	20·375	4	1	...	...	1					
	19·875	1									
	19·375										
	18·875										
	18·375	1									
	17·875										
	17·375										
	16·875										
	16·375	1									

TABLE 5.

Mental age in years	Body weight	$\sqrt[3]{}$ Body weight
1	76·0— 87·9	91·0— 95·7
3	92·4— 99·8	97·6—100·2
4	98·4—108·1	99·6—102·2
5	95·0—106·1	98·3—102·2
6	102·3—112·5	100·9—104·3
7	96·8—106·6	99·2—102·4
8	98·6—112·1	99·6—104·1
9	103·5—121·0	101·4—106·7
10	88·6—116·4	96·2—105·6
11	91·5—143·2	97·7—113·7

Mental age in years	Arm length	Foot length
1	92·1— 97·1	90·8— 96·1
3	97·9—100·9	98·1—101·8
4	97·0—101·1	96·8—101·2
5	97·0—101·5	98·8—102·9
6	99·9—104·9	98·3—104·5
7	100·1—104·4	98·7—104·9
8	100·5—106·1	99·6—104·3
9	99·6—105·4	101·4—107·8
10	95·1—107·8	94·7—107·2
11	100·7—114·0	102·6—111·9

APPENDIX B.

The measurements are divisible into two natural groups.—We calculate, by the method of least squares, the slopes b of the various regression lines of the standardized measurements on mental age. We also calculate the variances of these estimates. If p is any particular measurement, we find the following:—

TABLE 6.

p	Measurement	b	V_b	n
1	Head length	+0·5770	0·014732	269
2	Head breadth	+0·4012	0·010908	,,
3	Ear to ear	+0·5169	0·013770	,,
4	$\sqrt[3]{}$ brain volume... ...	+0·5281	0·010822	,,
5	$\sqrt[3]{}$ body weight	+0·9845	0·017126	,,
6	Arm length	+0·9698	0·021852	256
7	Foot length	+1·0273	0·026423	,,

By inspection we see that the values of b fall into two groups: (1) around 0·5 and (2) around 1·0. We may test the significance of this separation in the following way:—

If the seven values of b are really derived from the same material, the differences will be due solely to random sampling, and $\sum_{p=1}^{7} \frac{(b_p - \bar{b})^2}{V_p}$ will be distributed as is χ^2 for n = 6. Actually we find $\chi^2 = 27{\cdot}30$ and P is less than 0·01. Clearly the b's are not homogeneous.

If the data really consist of two separate parts, $p = 1$ to 4, and $p = 5$ to 7, then if b_1 = mean b from 1 to 4, $\sum_{p=1}^{4} \frac{(b_p - \bar{b}_1)^2}{V_p} + \sum_{p=5}^{7} \frac{(b_p - \bar{b}_2)^2}{V_p}$ will be distributed as is χ^2 for n = 5. We find $\chi^2 = 1{\cdot}48$ and P = 0·92. The discrepancy has completely vanished, showing that seven values of b must be divided into two groups, and showing that each sub-group is apparently homogeneous.

APPENDIX C.

Combining the Measurements.—We may combine the data simply by finding the general mean at each mental age. To find the variance of these combined means is not so simple. To take group A, the variance of the sum of four quantities is equal to the sum of the four variances plus twice the

product $r_{st}\sqrt{(V_s V_t)}$, the latter being applied to all the possible pairs ($\frac{n(n-1)}{2}$ in number) and summed. This latter part, involving the correlations between all the data in pairs, involves much computation. Since, however, the variances are all practically equal (apart from sampling variations) and as the correlations will all be about $+0{\cdot}5$, and as we are not going to apply further tests, we may simplify the latter to the expression, for the variance of the mean of n quantities,

$$V \text{ (grand mean)} = \frac{n + 1}{2n^2} \text{ (sum of variances of means).}$$

On examining the data in group A, we find that the variances of head height are consistently above the others, and we find, by testing, that the variances of our final (combined) estimates are actually larger when head height is included than when it is left out. Head height is thus not sufficiently precise to ' pay its way,' and its inclusion, instead of adding to the final precision, actually lowers it. In group A, we have therefore omitted head height when combining them.

By following exactly the same lines as before, we find the limits of the means to be:—

TABLE 7.

		Group A	Group B
Mental age in years	1	95·8— 98·5	91·7— 95·8
	3	98·0—100·1	98·1—100·7
	4	99·3—101·8	98·2—101·3
	5	98·3—101·1	98·4—101·8
	6	99·2—102·5	100·2—104·1
	7	100·0—102·4	99·7—103·5
	8	100·5—102·5	100·4—104·3
	9	99·6—102·1	101·5—106·0
	10	99·4—105·7	97·1—105·1
	11	100·1—105·1	103·4—110·2

THE MENTAL SYMPTOMS ASSOCIATED WITH 58 CASES OF CEREBRAL TUMOUR.

By

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In this paper a study of the mental symptoms found in cases of cerebral tumour was undertaken, and an attempt made to assess their value, if any, in the localization of the tumours. Concurrently with these symptoms, the presence or absence of physical signs was noted with a view to helping in localization.

The type of case admitted to the Maudsley Hospital must obviously differ from that referred to a general or neurological hospital, as those showing neurological signs only, or signs combined with very slight mental disturbance, would be referred to the latter, whereas those with marked mental disturbance would be referred to the Maudsley Hospital. Many cases suffering from aphasia were sent here as suffering from mental disorder, the apparently rambling and disconnected talk of the aphasic patient having been interpreted as gross mental disturbance.

In order to compare the cases admitted to the two types of hospital the records of a similar number of cases of cerebral tumour admitted to th Maida Vale Hospital were examined, and the comparisons will be given later.

Fifty-eight patients were examined. The localization of the tumours can be tabulated as follows:—

Left side	Right side	Mid-line or posterior fossa
22 frontal	4 frontal	5 posterior fossa
12 temporal	3 temporal	2 corpus callosum
4 parietal	3 parietal	1 pineal
	1 motor area	1 thalamus
—	—	—
38	11	9
—	—	—

It will be seen that of the 58, 49 (or 84 per cent.) were in the cerebrum, and only 5, or 9 per cent. were subtentorial. This would suggest that mental symptoms are associated much less frequently with subtentorial growths,

which were referred on account of the predominance of neurological signs to other hospitals. Mental changes were not observed until the intracranial tension increased sufficiently to produce somnolence and apathy, which progressed to coma. Thirty-eight were on the left side; it would appear that mental changes are more common with left-sided growths than those on the right side. Kolodny[1] appears to think they are commoner in left-sided tumours as a result of the associated speech disturbances. He states that as speech depends on visual and auditory impressions, it follows that if these are not fully understood they will not be so well retained, and thus memory impairment occurs. It may well be that detailed psychiatric examination is extremely difficult in aphasic patients and that an apparent memory defect is present as a result. There does not appear to be any doubt, however, that in all cases presenting aphasia there appears to be some reduction of intellectual capacity per se.

That mental symptoms are more common with cortical lesions will also be agreed, and when they occur with tumours in other situations they appear to be the result of increased intracranial pressure.

STRESS.

In all patients admitted with organic lesions, a detailed history was taken as in ordinary psychiatric cases, and an interesting point emerged, viz. that in many cases the relatives described the change following physical or mental stress.

Of the 58 cases examined, 19 were definitely (according to the history) associated with the following types of stress:—

Accident	4
Unemployment	2
Financial worry	2
Illness and deaths of relatives	3
Dermatitis, influenza, pregnancy	3
Overwork, love affairs and religious worry	5

It may be argued that the stress first caused the symptoms, which were already present, to be noticed, and this may hold good in some cases; but in others, however, close questioning elicited the fact that no change was noticed previously to the stress.

Thus one man, previously in good health, who worked as a guard on the underground, saw a girl attempting to board a moving train, and she slipped between it and the platform. He rushed to help her, and she regained the platform unhurt. He was very much shaken by the incident, but managed to finish his day's work. On returning home he attempted to write a letter to his brother, but was unable to do so as his hand felt weak (he was right-handed). The next day his right arm and leg were paralysed, and he became

aphasic. He was admitted to hospital but became comatose and died. Post-mortem examination revealed a glioma with a recent hæmorrhage into it.

In another case the patient sustained an injury to his arm and began to worry lest he should become paralyzed like his father. He became depressed, and attempted to commit suicide. His illness was noted by his friends as dating from this accident, prior to which he had been quite well. Post-mortem a tumour of the corpus callosum was found.

In all the other cases where stress appeared to determine the illness, the history given was like that already described (the latter), the onset and progress being insidious, as opposed to the first one, where the symptoms appeared in a dramatic and sudden manner. The part played by the stress, if any, will be discussed later in the paper.

MENTAL CHANGES.

For the purposes of description the mental changes have been divided into four groups; but it is obvious in most cases that groups I and II cannot be absolutely separated, as in group I a mild degree of intellectual impairment was necessarily present and in group II changes in emotional tone were found, although slight.

Group I.	Simulating Functional Psychoses.	
	States of depression	14
	States of excitement	7
	Schizophrenic state	1
	Anxiety state	1
	Obsessional state	1
	Hysteria	1
Group II.	Those with marked organic mental changes without clouding of consciousness	23
Group III.	Those with marked organic mental changes with clouding of consciousness	6
Group IV.	Hallucinations (without other psychotic manifestations)	4

Group I.—1. States of depression.—The localization of the tumours in this group was as follows:—

Right temporal	2
Left frontal	8
Right frontal	1
Corpus callosum	1
Cerebellum	1
Right motor area	1

It will be seen that the distribution of the growths varied considerably, and that nine out of the 14 were in the frontal lobe (eight, left; and one, right).

An interesting point is, that all these patients had long histories before admission, the average length of illness being five years, the longest being 12 years and the shortest one and a half years. Six of the patients showed typical depressive states, indistinguishable from an ordinary reactive depression without any intellectual impairment whatever. Thus one patient, age 64, became agitated, depressed and worried. He was self-reproachful, had ideas of reference, heard accusatory voices and thought he was to be killed. Memory and orientation were good. He showed no physical signs, localizing or general. He eventually died, and post-mortem a right temporal lobe tumour was found.

Another patient, age 64, who had an attack of depression 25 years ago from which he completely recovered, became depressed, restless and agitated. He was self-reproachful over imaginary wrongs and expressed persecutory ideas. Consciousness was clear and no physical signs were present. This depression followed a recurrence of a thyroid carcinoma removed by operation. The patient died, and a secondary deposit was found in the right temporal lobe.

These are two typical instances of depressive attacks occurring with cerebral tumour.

The other eight patients did not show such typical states as the above, but rather tended to show an exaggeration of their normal depressive tendencies. All these patients were described by their relatives as being of a quiet disposition, somewhat anxious, pessimistic in outlook and inclined to worry.

An interesting case in this group was that of a schoolmaster of education, who had always shown homosexual characteristics in his ' make-up.' He avoided women, preferred men's company, and had always taken interest in boys' figures and masturbation. In addition he had always been preoccupied with his bowels, and on his marriage, late in life, there were sexual difficulties and no relations with his wife. He developed a slow-growing astrocytoma of the left frontal lobe and gradually became more overt in his homosexual practices. He talked openly of sodomy, masturbated freely with boys, and became addicted to bouts of drunkenness. He clamoured for homosexual relations and eventually projected these on to his environment and accused people of practising homosexuality and of keeping boys in the ward for that purpose.

The marked feature in all these cases was the gradual increase in the depressive tendencies already noted in the patients' ' make-up.' They tended to become anxious and apprehensive, were extremely introspective and worried about their bodily health, and in some cases, became self-absorbed and apathetic, whilst in others they were agitated, fidgety and restless.

2. States of excitement.—Of the seven patients showing excitement, these had all been described by their relatives as being of a jolly disposition,

looking on the bright side of life and always fond of a joke. The distribution of the tumours was as follows:—

Left frontal	...	...	...	3
Right frontal	...	...	...	2
Left temporal	...	...	...	2

After the development of the tumour, these patients showed a gradual increase in their normal tendencies. They became facetious; treated their illness with undue levity; joked and punned and were amused at any trivial occurrence. They showed no insight into the severity of their condition but were elated and definitely euphoric, stating they had never felt better.

This state, sometimes called ' moria,' sometimes ' Witzelsucht,' which has been ascribed by some writers to lesions of the frontal lobes, appears to depend on the patients' ' make-up ' rather than on any particular situation of the tumour.

One patient in this group was of particular interest, in that slight changes in his personality had been noted over 18 months, but nothing definite was seen until three weeks before admission, when he became extravagant, embarked on wild schemes, bought villas and lost all his money. On examination he was aphasic; his memory was grossly impaired and he was disorientated. There were no physical signs except aphasia. His case appeared to be an organic one with expansive changes.

Spinal fluid examination showed:—Lange 5555432110; protein ·110 mgm.; cells 2 per c.mm.; Wassermann reaction, negative.

The patient died, and post-mortem a left temporal growth was found.

In all the cases in this group the chief change was the increase in tendencies already present, with some slight intellectual impairment. This type of change appears to occur in slow-growing tumours, and to be independent of the situation of the tumour. Emotional lability was noted, although the mood seemed fairly constant, and showed slight and short variations similar to those occurring in ordinary manic states.

3. THE SCHIZOPHRENIC REACTION was seen in a patient with a pineal tumour. She had shown physical signs suggestive of pineal tumour for some four years, but no mental dysfunction until a few days before admission here. She then was excited, restless, noisy and impulsive. She talked in a disjointed manner; grimaced; was negativistic and resistive. In addition she was aurally hallucinated.

After four months she recovered completely, and showed complete mental normality, but physical signs such as optic atrophy, sluggish pupils, defective upward and downward movement of the eyes, etc., persisted. Handley[2] states that confusion and agitation associated with hallucinations are found with pineal tumours, but it seems more probable in this case that the schizophrenic illness was purely intercurrent, and occurred in a patient

with a pineal tumour; upon it the latter had no bearing. About 18 months after leaving the hospital, the patient remains well mentally.

4. THE ANXIETY STATE occurred in a woman of 45 who had an attack of anxiety about the age of 20, from which she recovered, and who remained quite well until the air raids of the War. She then became apprehensive and jumpy, and began to sleep badly. At the same time her brother died and there was a marked increase in the symptoms. She was somewhat depressed, felt anxious and afraid of something indefinite. At times she was giddy, had marked anxiety dreams about being chased and murdered, and sweated profusely. Tachycardia was present, exaggerated deep reflexes and some enlargement of the thyroid also. There was no impairment of memory, and neurological signs were absent. These symptoms persisted, and post-mortem examination showed a right frontal growth.

5. OBSESSIONAL FEATURES occurred in a patient of 55 who began to have generalized convulsions, and who, just preceding the fit, had to count five, while the head felt as if it were being twisted round. There were no obsessive features in the personality previously, and none were found between the attacks. No physical signs were present. The patient died from a left frontal growth.

6. HYSTERICAL FEATURES occurred in a girl of 23 who had previously been in good health, and shown no neurotic traits except somnambulism. Six weeks prior to her illness her family made her break off her engagement against her wish, and this upset her considerably. She became mildly depressed and apathetic, refused food and suffered from anorexia and vomiting. There was no impairment of memory but vague physical signs were present. The day after admission patient became unconscious and died. Post-mortem a large glioma of the right parietal lobe was found.

GROUP II.—THOSE WITH MARKED ORGANIC MENTAL CHANGES WITHOUT CLOUDING OF CONSCIOUSNESS.—In this group there were 23 patients with tumours localized as follows:—

Left frontal	9
,, temporal	7
,, parietal	4
Right parietal	1
,, temporal	1
Thalamic	1

It will be seen that left-sided tumours predominated, viz., 20 out of 23, and of these nine were frontal and seven temporal.

The average history given was shorter than in the ' functional ' group already described, but longer than in the delirious group. An early feature was irritability with easy fatigability. The former appeared to be due to the failure in memory and the associated difficulty in coping with daily

routine matters. The fatigability was marked in testing memory, where in the early stages of the examination answers were given correctly and fairly rapidly, but as it proceeded they were given only after long intervals, and were often incorrect. Memory showed the usual impairment noted in all organic brain diseases. Recent impressions were lost first (retention) and in the later stages remote memory was impaired also. Confabulations, used to fill in the gaps in memory, were uncommon in this series and were only noted in one patient with a right temporal tumour. The intellectual functions at the same time became disturbed; thus judgment, discrimination and the critical faculty were markedly impaired. Coincidently, speech became slower and retarded, and in some cases the patient broke off in the middle of sentences owing to his inability to concentrate and his forgetting what he was saying. Perseveration was present in 12 patients with left frontal (7), left temporal (4) and left parietal (1) tumours. Orientation became disturbed in the later stages, firstly in time and space and finally for self. Coupled with the irritability, impatience, querulousness and outbursts of anger were sometimes present and restlessness was marked.

As the intracranial pressure increased, the patients passed into a somnolent apathetic state from which they could be roused and made to eat, and answer questions appropriately, although slowly. Marked variability is often present in this state, at one time the patient being somnolent and difficult to rouse, and at another, alert and well in touch with his surroundings. Emotional lability is often marked, the patient passing very readily from laughter to tears and with very little causation.

As the somnolence increased, external stimuli passed unnoticed, the patient having to be repeatedly urged to carry out any act; in this stage, incontinence of urine and fæces was common. Carelessness and self-neglect became very marked in the later stages.

GROUP III.—THOSE WITH MARKED ORGANIC MENTAL CHANGES WITH CLOUDING OF CONSCIOUSNESS.—Six patients presented symptoms as above. The localization of the tumours was as follows:—

Left temporal	3
,, frontal	1
Corpus callosum	1
Right parietal	1

All these patients gave comparatively short histories, viz., from six weeks to 12 months, the average being six months. Their illnesses were typical confusional states, and in the main were as follows.

They were completely disorientated; their memories were grossly impaired; conversation was rambling and incoherent. Identities were mistaken. They were noisy, restless and excited and showed great variability in their mood. At times they were suspicious and querulous, at

others depressed and apprehensive, whilst at others they were laughing and playful.

They expressed fleeting and ever changing delusions, e.g. that their spouses were unfaithful—that they were married to the doctors—that their food was being poisoned, etc. Hallucinations of both sight and hearing were present in all cases; the former took the form of flames and flashes, animals, costermongers, grotesque figures. The latter comprised bells, voices, bangs and shots. One patient (left temporal) in addition complained of a bad smell. Incontinence of both urine and fæces was common; and some were resistive over food, requiring to be fed.

Five of these patients showed physical signs of cerebral tumour; in one (left temporal) they were absent. The patients were all over 45 years of age, and all died within a short period of admission, the tumours being verified post-mortem.

GROUP IV.—HALLUCINATIONS.—Under this heading are described those cases in which no psychotic phenomena, except hallucinations, were found, in spite of careful clinical examination. Those patients with clouding of consciousness showed visual and auditory hallucinations and some of the depressive ones auditory and olfactory hallucinations.

Two patients with left frontal tumours in a setting of clear consciousness smelt a bad smell all day, as did one other patient with a left temporal tumour (without any other psychotic symptoms being noted). One patient with a tumour of the corpus callosum, extending into the præcuneus, saw figures of unknown men and women moving across the visual fields with her eyes closed, but not when they were open. No other patients with hallucinations as isolated mental symptoms were found in the present series.

APHASIA.

Twenty-three of the patients showed some degree of aphasia on admission, and in all cases the aphasia was a combination of motor and sensory varieties. It varied from a mild degree where the patient used a wrong word occasionally, and recognized the fact, to an unintelligible jargon, when newly coined words were used and the conversation was a mere incoherent hotch-potch. It was the latter type of patients who were described by their own doctors as being ' mental,' and whose condition was not recognized as a true aphasia. In most cases the aphasia was increased by emotional upset such as excitement of any kind, and in a few cases was present only preceding or following a generalized convulsion.

It is a debatable point whether the intellect is impaired in aphasia. Many writers state that aphasic symptoms are due to intellectual impairment, as in some cases of severe dementia aphasia is not present, and in some cases of aphasia little intellectual impairment can be found. Marie[3] affirms that

inability to understand impressions, or to interpret read or spoken thoughts, must be looked upon as intellectual impairment. It is often advanced, however, that there is frequently no proportional relation between the severe aberrations of judgement, attention and memory on the one hand, and the aphasia on the other; and conversely, between the considerable aphasic disturbances and the intellectual defect.

The investigation of the intelligence of aphasic patients is extremely difficult, but probably with aphasic disturbances intellectual impairment in some degree occurs as well.

FITS.

Twenty-four of the patients admitted gave a history of fits.

Left Frontal—11.—Four of these were described as ' fainting attacks ' in which the patient became pale and pulseless without loss of consciousness. The patients were giddy and were said to ' lose themselves,' i.e., to lose the thread of the conversation momentarily. These appeared to be similar in some respects to petit mal, and after the attack the patient for a short time had difficulty in getting out his words and appeared to be dazed. In the remaining cases, in the earlier stages, the relatives described the patient as having attacks similar to the above, in which he ' lost himself ' and forgot what he was doing. In the later stages the fits took the form of generalized convulsions with loss of consciousness. In no case was a cry noted, and in only two was the tongue bitten. Incontinence of urine occurred in all, but in none was a history of incontinence of fæces obtained. The duration of the fits varied from two minutes to two hours, the average being roughly 15 minutes. Tonic and clonic stages were well marked and conjugate deviation of the eyes was present in three cases. After the attacks, three patients had difficulty in getting their words out, and were unable to articulate distinctly; one had a period of amnesia lasting from two to three hours, and another had a temporary paresis of the right leg with an extensor plantar response.

Left Temporal—5.—One patient had attacks which started in the right hand and spread up the arm, consciousness then being lost. The right leg was then drawn up and the cheeks blown out. These attacks lasted five minutes, and afterwards he was able to continue his work and felt no ill effects.

Another patient had ' turns ' lasting ten minutes, during which headache became very severe, and he was unable to speak. They recurred with great frequency but left no ill effects after they had passed off. Another patient had attacks characterized by weakness of the right hand and inability to find the right word. They lasted a few minutes and there was no loss of consciousness. Another patient had attacks of generalized shivering with no loss of consciousness. One patient had typical epileptic seizures which were treated as idiopathic epilepsy for some time.

Left Parietal—4.—One patient had a generalized convulsion with loss of consciousness, lasting ten minutes, this being preceded by black spots in front of the eyes. Another had a sensory Jacksonian fit beginning in the right fingers and showing the definite march up the arm and down side, thigh, leg and foot. Twitching then began in the toes and the patient felt as if someone was pushing her and she fell unconscious to the ground. She remained unconscious for 30 minutes. One patient had fainting attacks similar to those described under the heading of frontal tumours, and one had screaming attacks resembling an acute anxiety attack, in which he felt 'something jump from his ankle to his shoulder' and felt he was going to die.

Right Frontal—2.—One patient had attacks two to three times a day in which the face went blue and he trembled; there was a feeling of nausea and generalized trembling. The attack was only momentary and consciousness was not lost. The other patient had generalized convulsions indistinguishable from the major attacks of idiopathic epilepsy.

Corpus Callosum—1.—This patient had attacks lasting two days and at intervals of two weeks, in which there were headache, vomiting and giddiness, and pseudo-hallucinations of unknown figures seen only when the eyes were closed.

Cerebellar—1.—This patient had attacks in which the face blanched, and then the whole body rotated in a laying position with extraordinary rapidity from right to left, and ended in a semiprone position. The right upper limb was semi-flexed and a few clonic spasms occurred. The whole trunk and limbs then became flaccid and consciousness was lost. The sphincters were not relaxed and the eyes were directed downwards with the visual axes parallel. These attacks lasted three minutes, and were followed by periods of transient confusion lasting some hours. Following on the confusion were periods of depression with marked suicidal tendencies.

INCONTINENCE OF URINE AND FÆCES.

Incontinence of urine and fæces occurred in nine patients as follows:—

Left frontal	5
,, temporal	2
,, parietal	1
Right frontal	1

Incontinence of urine only occurred in:—

Left frontal	7
Right frontal	1
Left temporal	1
Right parietal	1

Incontinence of fæces only did not occur in any case.

Patients who showing clouding of consciousness, or were comatose, were excluded, as incontinence might obviously occur independently of the situation of the tumour.

In all the above cases the patients realized they had been incontinent and were quite indifferent to the fact. They were conscious of the stimulus but owing to their apathy did not make any attempt to control their sphincters.

OTHER PHYSICAL SIGNS.

Twenty-two patients of the 58 showed localizing signs, 24 showed vague neurological signs and in 12 physical signs were completely absent on admission.

Papillœdema was present in 35 of the patients on admission and was equal on the two sides in 23, was greater on the side of the lesion in five, and greater on the side opposite the lesion in seven.

An analysis of the headache, vomiting and papillœdema showed that in eight cases (two left frontal, one left temporal, two right temporal, one callosal, one cerebellar, and one right rolandic) none of these was present at any time.

DISCUSSION.

Stress.—One method by means of which stress can produce symptoms appears to be as follows. As is well known, sudden emotional disturbance causes a rise in blood-pressure, and this may be sufficiently high to cause one of the newly formed weak-walled vessels in the growth to give way, producing marked symptoms suddenly, or an increase in those already present. In those cases where slight symptoms are already present but are not recognized by patient or relatives, stress may possibly operate in the following manner. It is known that a psychological stimulus in the form of emotion may cause physiological disturbance. This is seen in the increase of spasticity in disseminated sclerosis and of the tremor and akinesis in encephalitis under intense emotional upset. The psychological stimulus may also act in this manner in cerebral tumours, thus causing the symptoms to be noticed objectively. Lastly, the organic lesion already present must necessarily make the patient's power of adaptability to any additional worry or shock less than normal, and provided he can carry on with his daily routine without undue stress nothing abnormal is noted. As soon, however, as he has to face any additional stress, he is unable to cope with it, and symptoms become more pronounced in that he is unable to carry on with his daily duties, which previously he did quite well.

Personality Changes.—In the patients with slow-growing tumours, irrespective of their localization, it has been shown that an increase in preformed personality characteristics was marked and intellectual disturb-

ances slight. It would appear then, that there is a release of these tendencies, or that the normal inhibition is removed. Attempting to describe this on Hughlings Jackson's principles it might be suggested that these are positive release symptoms comparable to positive neurological release symptoms such as fits, spasticity, etc.; the negative mental symptoms —memory impairment, defect of attention, etc.—corresponding to negative neurological symptoms such as paralysis, anæsthesia, etc.

Byrom Bramwell[4] in 1888 described the mental changes associated with cerebral tumours in this manner. He includes among the positive symptoms ' irritability and unevenness of temper, crossness, oddness of manner and address, etc.,' and the negative symptoms ' intellectual impairment, loss of memory, want of attention, loss of power of mental application, etc.' Gordon Holmes[5] states that both depression and euphoria may be found in frontal lobe tumours, the former with incontinence, drowsiness and stupor, and the latter with restlessness, exuberance, childishness and facetiousnesss. Here again it would appear that the emotional tone depends on the previous personality of the patient.

Henry[6], in the most recent article on this subject, appears to think that in all cases of cerebral tumour the first prevailing mood is depression, due to consciousness of failing ability to work and cope with things in general. The next change, according to him, is one of euphoria, which is a means of escape from suffering, or other avenues of escape are provided by a delirious reaction or somnolent state. In the present series, although variability of mood was found, the patient was either predominantly euphoric or depressed from the beginning of the illness. In those cases showing euphoria there was no initial period of depression; hence it would appear that the mood depended on the previous ' make-up ' of the patient, and not on his reaction to the physical illness. In no case was there a striking change from a depressive to a euphoric state as the disease progressed.

Kolodny[7] found that changes in personality were present in 21 per cent. of temporal tumours as against 5 per cent. of frontal tumours. In this series, of the 21 cases showing personality changes, 14 were frontal (54 per cent. of the frontal tumours examined) and four were temporal (27 per cent. of the temporal tumours). Localization would appear, therefore, to have little bearing on these changes; rather, as already stated, the tumour tends to release preformed tendencies.

Depressive states were commoner in this series, and this is in keeping with the general admissions to the Maudsley Hospital, in that depressive states are much commoner than manic ones.

Intellectual Impairment, etc.—Kolodny states that 19 of 38 cases of temporal tumour showed mental impairment and only 13 of 30 frontal. In this series nine frontal and seven temporal showed mental deterioration in a setting of clear consciousness, i.e. 35 per cent. and 46 per cent. respectively.

It seems, however, that these changes depend rather on the rate of growth than on the site. The slowly growing ones produce personality changes, and the more rapidly growing ones the changes in the intellectual sphere.

It may be that some disturbance in the circulation of the cerebrospinal fluid causes the symptoms, and in the former there is time for the circulation to adjust itself, whereas in the latter there is not.

General Neurological Signs.—A striking fact was the paucity of physical signs in many of the cases admitted. Twelve showed no physical signs, 24 showed only vague ones and only 22 (38 per cent.) showed localizing signs. Comparing this with cases admitted to Maida Vale, 36 were cerebral and 19 subtentorial in contrast to 49 cerebral and 5 subtentorial admitted to the Maudsley. There was no great discrepancy in generalized signs, but localizing signs were very much commoner. Thus, 51 showed localizing signs, in seven they were vague, and in none absent.

Three cases were hallucinated; in two of these visual hallucinations were present in a hemianopic field, and in the third (a case of cerebellar tumour) flashes of light occurred.

Twenty-three of the patients admitted to the Maudsley showed aphasia, and only 12 of those admitted to Maida Vale. Only 10 of those admitted to Maida Vale showed mental symptoms and then only mild depression, impairment of memory, irritability, or visual hallucinations in a hemianopic field; and none simulated functional states such as is the case in the present series. One patient with a right frontal tumour was cheerful and jocose and was said to have always been so, and another who was always suspicious and jealous had shown an increase in these tendencies since the development of a left middle fossa tumour.

SUMMARY AND CONCLUSIONS.

1. Mental symptoms in cerebral tumour do not appear to depend on localization, but rather on the rate of growth of the tumour and the previous personality of the patient.

2. Mental symptoms occur more commonly with cerebral tumours (more with left- than right-sided ones), as, of the 58 examined, 49 were cerebral and only five subtentorial. All the former showed mental symptoms and only one of the latter.

3. In slow-growing tumours personality changes predominate, the mood depending on the release of preformed tendencies. In this series, 14 of 58 examined were depressive and seven manic.

4. In rapidly growing tumours changes in the intellectual sphere are marked and personality changes slight, while in the more rapidly growing tumours intellectual disturbances with clouded consciousness are found. In the former group were 23, and in the latter six.

5. Twenty-five cases showed reactions simulating functional psychoses, and in 12 of these physical signs were absent.

6. Physical signs are less common in patients admitted to a hospital of the Maudsley type than in patients admitted to a neurological hospital, whilst aphasia is commoner in patients seen in the former.

I wish to thank Dr. Edward Mapother for permission to make use of the clinical material at the Maudsley Hospital, and I am indebted to the staff of the Hospital for Paralysis and Epilepsy, Maida Vale, for use of their records.

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Short Notes and Clinical Cases.

CONGENITAL DEFICIENCY OF CONSCIOUS AUDITORY PERCEPTION OF WORDS (WORD-DEAFNESS):

WITH REMARKS ON OTHER DEFICIENCIES OF CONSCIOUS PERCEPTION.

By

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The patient, an English boy (J. B.)[1], age 3 years, is of at least average intelligence and general development, excepting for the defect which I should prefer to call 'congenital deficiency of conscious auditory perception of words,' generally known as word-deafness or 'surditas verbalis.' By analogy I should prefer to call the corresponding condition of word-blindness ('cæcitas verbalis') 'congenital deficiency of conscious visual perception of words.'[2]

The only words the boy can utter are 'da-da' and 'ma-ma,' but he cries and laughs in the normal way with appropriate facial expression, and expresses his satisfaction or interest or excitement by ordinary infantile sounds, which are rather musical, not loud, and never disagreeable to hear. He can call attention to or obtain what he wants by pointing and other gestures accompanied by sounds. He can certainly hear *loud noises*, for his attention can be attracted by the ringing of a bell, clapping of hands, or shouting, but he seems to react rather sluggishly, and there is no palpebral reaction to an ordinary coin-click. He does not seem particularly to like music (gramophone or 'wireless'). He is normally interested with everything he sees and from a picture-book he can pick out pictures of animals that he wishes to show to his parents. He can play ordinary games or dance with other children or with adults, is good-tempered, and never mischievous, excepting unintentionally at times owing to his great curiosity for intelligently examining everything. He apparently has occasionally said 'ta' on being given something, but ordinarily when wanted to thank anyone, only moves his lips, as if it was that he was wanted to do. He sometimes wets his bed at night, but in the day-time always makes it understood that he

[1] The case was demonstrated at the meeting of the Royal Society of Medicine, Section for the Study of Disease in Children, on January 27, 1933.

[2] Cf. F. P. Weber, 'Case of Congenital Word-Blindness,' *Brit. Journ. Child. Dis.*, 1917, xiv, 183.

wishes to go to the closet. When he comes home to his mother he will let her know in his own way what he has seen and experienced—for instance, using a little hammer to show that his knee-jerks have been examined at the hospital. When the baker rings the bell he will point to where the baker's book is lying, and he helps his grandmother in getting the spoons, etc., ready for tea. Although, of course, he cannot lip-read, he always somehow understands what his grandmother tells him to do—doubtless by her facial expression and gestures.

The boy is well-built, and nothing abnormal is found in his thorax, abdomen, limbs or urine. His pupils react to light, and his knee-jerks are normal. Nothing abnormal was seen by ophthalmoscopic examination (Dr. C. Markus). Dr. W. Wilson finds the mouth, fauces and ears (otoscopic examination) normal, and there is a normal response to the caloric test (cold syringing—left ear only tried). There is no history of any trauma or serious infection. His mother was in a motor-car smash when she was in the third month of pregnancy. The blood-serums of the father and mother both give negative Wassermann and Meinicke reactions. The child prefers to use his left hand, as his maternal grandmother did in childhood. He is the only child (result of the only pregnancy), as his mother was. Both father and mother enjoy good health. The father is one of a family of nineteen brothers and two sisters. There is no history of deafness, dumbness or abnormality of speech in the relatives, or of delay in learning to speak, imbecility, insanity, epilepsy, tuberculosis or any special tendency to shortness of life. There is no consanguinity between the patient's parents.

REMARKS ON OTHER DEFICIENCIES OF CONSCIOUS PERCEPTION.

In regard to the neurological and other problems connected with the subject and for an analysis of the literature, see C. Worster-Drought and I. M. Allen, this JOURNAL, 1929, IX, pp. 193, 289, and 1930, X, p. 193. These authors prefer to call the condition ' congenital auditory imperception,' and especially discuss its relation to idioglossia and other speech defects. In the present case it is of course doubtful how far the patient can consciously perceive sounds (musical, etc.) other than those connected with language. I will not enter here into all the questions of defective conscious auditory and visual perceptions apart from perception concerned with language.

In the heading I have written ' conscious auditory perception ' intentionally, to show that I believe there are also *subconscious* and *unconscious*, and not only *conscious*, auditory (and, of course, other) perceptions. I admit that my terminology in this respect involves enlarging the definition of the word ' perception ' so as to include what is manifested when a response of any kind follows any kind of sensory stimulus, no matter what the level in the central nervous system may be of the path between the stimulus and the resulting response or reaction. ' Perception,' in this

use of the term, may be mindless (or unconscious) as well as mental (or conscious); for convenience of terminology I postulate that perception is present (apart from any percipient mind) whenever any congenital (inherited) or acquired (conditioned) reflex action takes place.

Analogous to congenital-developmental defects in the conscious perception of words, congenital-developmental defects in the conscious perception of smell and taste probably likewise occur, but they must be even rarer, for doubtless the conscious perception of smell and the conscious perception of taste are almost as primitive as conscious perceptions of touch and pain, and are therefore extremely unlikely to be congenitally wanting or to be so weak that, like any *locus minoris resistentiæ*, they are easily lost (temporarily or permanently) from various post-natal causes. Moreover, such congenital-developmental defects in smell and taste would readily escape recognition (as partial or complete colour-blindness often does, though this is not strictly analogous).

In cases of congenital dumbness or ' congenital aphasia ' of the above-described kind (congenital word-deafness) it is known that the patients subsequently learn to speak—with or without marked ' idioglossia.' Adaptation in brain defects may certainly (as is well known) be wonderful, and together with adaptation the natural post-natal development of the cerebral cortex must be taken into consideration. A question arises whether in similar cases the actual commencement of speech may ever be very sudden. One could conceive that a gradual adaptive development might silently take place in the brain to such an extent that under the influence of some shock, powerful emotion or danger, speech might suddenly spring forth from a hitherto dumb child like fully-armed Athene from the head of Zeus.

A few doubtful examples to illustrate this idea have been recorded in modern times, but by far the most interesting is one considerably over two thousand years ago, namely, the oft-repeated story narrated by Herodotus, the ' father of history.' At the battle of Pteria (B.C. 546), when Crœsus, King of Lydia, was overthrown by the Persian Cyrus, a soldier in the Persian army was about to kill Crœsus, not knowing who he was, when the latter's son, who had been dumb from birth, called out: ' Do not kill the King Crœsus.'

> One may explain this story in the way suggested above, instead of telling Herodotus to his ghostly face that he was uncritical in his acceptance of tales and sagas, as an English scholar somewhat irreverently put it:
>
> ' The priests of Egypt gammoned you;
> It was not very hard to do.
> We will not let you gammon us,
> Herodotus, you ancient cuss!'

For comparison with cases of deficiency in conscious perception of words, etc., I will here shortly refer to a case in which *all conscious perception for every kind of stimuli was permanently lost* after an operation under ether

anæsthesia, during which severe general convulsions occurred, succeeded by prolonged status convulsivus. It seemed as if the whole cerebral cortical substance concerned with conscious perception had been permanently destroyed—just as the whole hepatic parenchyma has sometimes undergone selective destruction as the result of chloroform anæsthesia[1]. The patient was an English boy (J. N.) born on February 17, 1927. When I saw him in 1931 he was four years old, and his general bodily development and state of nutrition were good, but he was completely ' mindless ' and a cerebral diplegic. Up to the age of two years he had been mentally and bodily normal and his strange condition had followed the above-mentioned operation (for appendicitis) on March 11, 1929. In 1931 I described the boy's neurological and mental condition as follows:—

> When quietly asleep with his eyes closed he seems normal. When awake and, as usual, lying on his back in bed with knees, elbows and fingers more or less flexed, he does not at first sight appear very abnormal. But all his movements, which are chiefly of his limbs and head, are automatic, not volitional, and the movements are seldom sufficient to change his position from one part of the bed to another. He never, even automatically, attempts to grasp any object, such as a key or watch, when dangled against his hands. He cannot speak, though he occasionally utters animal-like, but apparently meaningless sounds. His placid, expressionless face hardly alters at all. All emotional manifestations (smiling, laughing, crying) are completely absent. His eyes sometimes appear to follow an object close to him, and he sometimes turns his head away from a very strong light, but he certainly does not consciously see. He seems not consciously to hear any sounds. He cannot, when asleep, be awakened by any loud sound. Even when awake he does not usually respond to sounds, though when called he has occasionally been observed to move his head. Dr. William Wilson, our aural specialist, who has kindly examined the child, reports that ' no palpebral reflex occurs to any noise stimulus.' Dr. Wilson has made the interesting observation that in this child nystagmus occurs normally on trying the caloric test.
>
> Anyhow, one is justified in saying that the child cannot either see or hear in the ordinary conscious way. The same may be stated in regard to sensations of touch and pain. No special tests of taste or smell have been tried, but the parents state that the child does not swallow anything that they themselves think has a markedly disagreeable taste. He certainly does not feel anything in the ordinary conscious way. His reactions seem to be only automatic. Local contact with a hot object produces a slight response. He sometimes makes a noise in apparent response to a stimulus which to most persons is unpleasant—for instance, when the external auditory meatus is syringed out with cold water in order to try the above-mentioned caloric test. He occasionally coughs, though I have not observed this myself. Sometimes he swallows better than at other times, but when he is fed with a spoon the food often remains a little in the mouth till it is passed on by a reflexly evoked swallowing movement. There is no abnormal salivation. He of course passes urine and fæces in the bed under him.

[1] See F. Parkes Weber, ' Complete Mindlessness and Cerebral Diplegia, after Status Convulsivus associated with Ether Anæsthesia,' *Brit. Jour. Child. Dis.*, 1931, xxviii, 14-21.

Since the above notes were written the condition of the boy (now six years old) has remained precisely the same (for this information I have to thank Dr. J. Sangster Greig, Medical Superintendent of the Forest Gate Hospital).

It seems to me that intimately associated with the question of various defects in conscious perception, of congenital-developmental nature (including word-deafness and word-blindness) or resulting from traumatism (wounds, etc.) or organic disease (ischæmic, toxæmic or destructive) of various kinds, are still larger and obscurer questions of temporary defects in conscious perception (of many kinds and various degrees) associated with the different stages of somnambulism, hypnotism, hysteria, shock, sleep, the action of various drugs, and extraneous and autogenous toxins. One may instance especially the hysterical disturbances of sensation which may be induced by psychic causes and which may be removed (though perhaps often only temporarily) by electric and other shocks. But I certainly need not discuss the various possible explanations, such as temporary disassociation, disconnection or separation of cortical nerve-cells (retraction of cell-processes, etc.).

Critical Review.

THE MYOTONIC PUPIL: A CONTRIBUTION AND A CRITICAL REVIEW.

By

SMITH ELY JELLIFFE, New York.

In 1932 Adie offered a condensation of some previous studies and sought to erect a new syndrome, consisting chiefly of tonic pupils and absent tendon-reflexes, which should be thought of as benign and to be separated from tabes dorsalis.

In his first contribution, made the year previously, the chief features of this syndrome were outlined: 'I wish to draw attention to a benign symptomless disorder characterized by pupils which react on accommodation and not to light and by absent tendon-reflexes.' Inasmuch as five of six cases reported came under his observation in a few weeks, he thinks it cannot be rare, but because of the possibilities of confusion with tabes dorsalis it merits recognition. Six case-histories are then reported briefly. In commenting on the pupillary signs he calls attention to certain variations which justify his title 'Pseudo-Argyll Robertson pupils.' In citing an early report of a patient with myotonic pupil by Wilbrand and Saenger, an 'imperfect stationary tabes,' he says:—'This is the only case resembling my own that I have been able to find in the literature.' He then later finds further observations from Moore and from Morgan and Symonds relative to the problem, and discusses the essentially syphilitic or non-syphilitic etiology of true Argyll Robertson pupils.

In a further study more specifically related to this latter problem Adie returns to the subject and in a post-graduate lecture outlines more in detail the matter of pseudo-Argyll Robertson pupils, a term which his colleague, Kinnier Wilson, some ten years or more ago, called a 'horrid expression.' Whether Wilson meant as horrid the abbreviated 'pseudo-Argyll' or its fuller form does not appear.

In his later contribution (1932) he finds a number of other cases spoken of as tonic pupillary reaction cases with absent tendon reflexes, beginning with one reported by Nonne (1902) in which the loss of ankle-jerks was thought to be due to a diabetic neuritis; Marcus' case (1906) might be congenital syphilis; Oloff (2 cases) (1914), no comment; Jess (1914), only right ankle-jerk absent; Lersperger (1914), no details (possible tabes);

Axenfeld (1919), probably congenital tabes (blood once suspicious); Gehrcke (1921), tabes; Behr (1921), ankle-jerks only absent; Weill and Reys (1926), polyneuritis and rheumatic fever or influenza; Moore (1931), 2 cases; and then his own 6 (1931) and 8 more in this article in *Brain*.

In a still later paper (1932) Adie again reviews the situation, this time in an ophthalmological journal, and would advance to the position of a ' name ' for the disorder.

Adie's reprint of this paper and one by Guillain and Sigwald came to my desk the same day, and as each dealt with the specific problems here already broached as well as those of personal interest, I have been stimulated to call attention to some of my own observations which thoroughly justify Adie's belief that the situation ' must be common.' Whether one is correct in calling the syndrome a disease sui generis is doubtful.

Were my priority propensities in the ascendant I would call attention to early contributions of mine (1915–1921) in which the exact situation of modified Argyll Robertson pupils and lost tendon-reflexes, precisely as described by Adie, was reported and the danger of confusion with tabes accented in two cases of hypothyroidism. Inasmuch as I have seen both patients at times when they have relapsed from the use of their thyroid and have again had not only diminished tendon-reflexes and sluggish pupils (as they were called in my paper) but the actual tonic pupil on which Adie makes his differentiation, particularly in his paper in *Brain*, I have minded to make this short note.

I would also emphasize what I have already stated that in influenza, in encephalitis particularly, in diabetes and in alcoholic polyneuritis, as well as in hypothyroid states I have found these tonic pupils and lost tendon-reflexes. It is provisionally held to be chiefly a ' neuritic ' syndrome and is not in my opinion entitled to a separate name. The isolated pupillary phenomena I have also seen in a pineal tumour (referred to by Wilson in 1921), also in tuberculosis of the apex, in disturbance of the sympathetic in the neck, and tuberculous gland operations and in other situations. I am convinced it is by no means a rare anomaly, as Gordon Holmes, Gehrcke and others have also reported.

Adie offers further synonyms for the pupillary reaction, such as ' pupillotonia, myotonic reaction, tonic convergence reactions of pupils apparently inactive to light, Marcus' peculiar pupil phenomenon, non-luetic Argyll Robertson pupil, pseudo-Argyll pupil ' (see Bing and Franceschetti, l. c., p. 126); these are at times present and at other times atypical of the tonic pupil. In a rather laboured way he differentiates (A) a complete form in which one or both eyes show the tonic convergence reaction in a pupil apparently inactive to light, with absence or diminution of one or more of the tendon-reflexes; and (B) incomplete forms, with (1) tonic pupils alone, (2) atypical phases of the tonic pupils alone, (3) atypical phases of the tonic pupil with absence of reflexes, and (4) absent tendon-reflexes alone.

He has found a preponderance of females, and in a ratio of 4:1 only one eye is involved. In this paper he describes at length the fairly well known variations in pupillary contractility, paying special attention to that type of contracted non-light-reacting pupil which nevertheless dilates slightly in a dark room and then on exposure to light contracts even smaller than before and then enlarges a trifle. This may occur in one or both eyes. Another feature is no loss of accommodation reaction: also that these may or may not be immobile pupils to convergence. No mention is made either of the consensual light reaction, or of the sympathetic reaction to pinching of the cheek, nor do we find any notes made upon the acoustic pupillary reflexes.

As for the occurrence of various gradations in the Argyll Robertson pupil, especially that variant called by Saenger the ' myotonic pupil ' and the ' pseudo-Argyll Robertson ' pupil by Adie, even a cursory glimpse at ophthalmological and neurological literature seems to indicate that the fluctuations and modifications are so numerous that it is an arbitrary matter to make hard and fast distinctions. Even in the development of the classical tabetic pupil there are cases in which the pupil goes through a ' myotonic stage,' and the literature seems to indicate much the same in other neurological situations. Even extreme Argyll Robertson tabetic and paretic miotic pupils are not absolutely stiff when examined in a dark room with lenses (see Bing and others).

With a proper microscope and pupillometer these fine gradations are readily observed and recorded. As is well known, ' miosis ' for Bach is 2·5 mm., for Shirmer 2·0 mm., and for Uhthoff 1·5 mm. In much the same manner of scale—from fixed, to almost fixed, to the ' Erholungs '-pupil in the dark, to even lesser degrees of loss of light reflex, or prompt or less prompt reaction to cocaine or homatropine—common sense recognizes tendencies rather than strict mathematical confines. We find Adie's grades A and B, 1, 2, 3, 4, rather academic.

A cursory glance at this same ophthalmological and neurological literature also reveals a multiplicity of situations, in which the ' myotonic ' pupils, as of the type here under consideration, are to be found. Some are reported as in ' normal ' individuals; just as abolition of the knee-jerks is found in 1 to 2 per cent. of ' normal ' people. An industrious compiler undoubtedly will find a motley group of myotonic pupil cases. An initial search in Bach, in Bumke, in Behre, Best, Bing (see Schieck and Brückner, p. 126, for example) reveals scores of them. In diabetes, in polioencephalitis, poliomyelitis, influenza, cervical rib, injury to cervical plexus, thyroid operations, œsophageal operations, neck operations, apical tuberculosis, alcoholism, injury to skull, delayed apoplexy, the feebleminded, catatonics, syringomyelia, multiple sclerosis, aortic aneurysm, cervical pachymeningitis, Thomsen's disease, etc., they are found, so that Bach writing even 10 years

ago says he has observed the myotonic reaction ' often ' (p. 141) and that ' it possesses no diagnostic significance ' (p. 144).

About loss of tendon-reflexes the situation is not so clear-cut, but here also there are many non-syphilitic factors that can bring about loss or great diminution of tendon-reflexes. The literature is vast and need not be entered into here.

The combination is undoubtedly rarer, but it too I believe is not so rare, and I am convinced it is not a specific syndrome in the sense of Adie.

Looking through the case-histories quoted as related to the syndrome, one finds that Holmes alone refers to thyroid states. In two of his patients the thyroid was enlarged and in one there had been a history of hyperthyroidism in earlier life, but there are no observations on any result of such modified thyroid states, if any, even though it is well recognized that hypothyroidism not infrequently follows hyperthyroidism.

It would be an absurdity on my part to assume that Dr. Adie is not familiar with many of these facts. The patients whose histories he has presented were probably not postencephalitics, nor did they have mesencephalic brain tumours, nor possibly (?) any of the conditions here mentioned as giving rise to varying degrees of ' tonic ' pupils. The evidence presented in the short histories, however, does not exclude some of the more frequent—even if but transitory—causes for the appearance of such tonic pupils. Among these, for example, alcoholism, influenza, beginning psychoses, ear disorders, psychoneuroses, tuberculosis, hypothyroid states must be considered. But even if the examinations had been extremely meticulous still it would remain a doubtful issue whether one could erect a disease sui generis upon a type of syndrome both aspects of which are in reality of widespread occurrence—tonic pupils and diminished knee-jerks.

Further, in following out Guillain and Sigwald's interesting communication I note that several issues pertinent to Adie's papers are discussed. In the first place, while agreeing in part with Adie's conception that such cases of tonic pupils and absent tendon-reflexes are met with (they report two such in this present communication) they are of the opinion that the interpretations given by Adie are not satisfactory, particularly when he would ally his cases with the myotonic dystrophies, periodic palsies or myasthenia. This, however, brings up other possibilities for the syndrome. In discussing the areflexia aspects of the situation they also disagree as to the vegetative nerve implications, in view of the known areflexias of more distinctly neuritic origin. They also take up a number of points concerning the true Argyll Robertson pupil as occurring in other conditions than in syphilis, about which Adie makes the categorical statement, ' The true Argyll Robertson phenomenon is an infallible sign of syphilis '—a position from which Bing has retreated after stoutly maintaining it for years. Wilson has covered this situation in his 1921 paper and later contributions. Guillain

and Sigwald quote a number of their own observations which run counter to this statement of Adie's.

Finally, Guillain and Sigwald come to regard the syndrome as of a toxi-infective nature of unknown origin, whereas I would maintain the same position, except that in some instances the toxic or infective process is obvious —diabetes, influenza, alcohol, etc., and sometimes not so certain— hypothyroid state, etc.

Inasmuch as my contribution (1921) has escaped the search of Adie, and as an earlier paper (1914) dealing with one of these patients has only been published in short abstract, I feel justified in citing them again with additional notes on the case of L. T., whom I saw later in 1918 and have heard from since. The other patient has been lost sight of for some time.

PERSONAL CASES.

CASE I.—Mrs. X., first seen November, 1913, 54 years of age, twice married, at 19 and at 27. First husband died of tuberculosis. She had much economic worry. Two children of this marriage; second marriage four children. General neurotic history. No alcoholism and no history of abortions, or skin eruptions. Some thinning of her hair. Menopause at 45 with exaggeration of neurosis.

She came with several documents of previous examination from competent physicians and neurologists, one or two of whom diagnosed tabes dorsalis.

She had had neuralgic pains in the arms and legs almost crisis-like in their severity and mode of occurrence. There were three attacks of what might erroneously be taken for gastric crises in the two years before 1913.

Notes of examination, somwhat more fully reported than in the brief communications made in 1914 and 1921, showed a somewhat stout and heavy woman. The skin was thick, slightly pigmented, and dry and slightly scaly. It did not pit on pressure. She had noted she did not sweat much. The skin felt like marble to the touch. She was always chilly. The hair was thin and shedding, the eyebrows particularly were scanty, the nails fragile. The lips were somewhat thick; the mouth and slightly thickend tongue were dry, and the latter marked by the teeth. The abdomen was fat. The joints were painful and stiff. The pulse was slow; mostly about 54-60. She was constipated and slightly anæmic.

Neurologically: She was not taken up with her food and the nuances of smell were distinctly not interesting. The eyesight was unaltered. The fundus showed no marked anomalies. The pupils were unequal, the left 5 mm., the right 2 mm. There was a marked sluggish light reflex in both eyes which was more striking in the dark room. She had a typical myotonic pupil. The convergence reflex reaction was present. There was a definite sympathetic reflex to pinching the cheek but the consensual light reflex was even less present than the direct, but very minutely present in the right eye more than the left. There were no ocular palsies. The palpebral fissures were slightly unequal. No nystagmus. The functions of the fifth and seventh nerves were without marked anomalies. Hearing was defective. Rinné +, i.e., bone conduction was as good as air conduction. There was no marked defect in equilibration. Saliva was thick and ropey.

Upper extremities showed no atrophies, hypertrophies, anæsthesiæ or paralyses. There was some hypotonia and not over-active elbow and wrist

tendon-reflexes. No ataxia in finger-nose and finger-finger tests; no astereognosis, no bone sensibility changes; no thermal sensory defect.

Trunk: Abdominal reflexes sluggish (fat). Constipation: no bladder disturbances.

Lower extremities: As upper, but some nerve tenderness on pressure and more definite loss of tendon-reflexes, especially knee-jerk and achilles-reflex. Jendrassik's method reinforced the scarcely elicitable knee-jerks. No ataxia, no Romberg's sign; knee-heel test was normal. Bone conduction, discrimination, normal. Position sense normal. No heat or cold losses, nor fine sensibility changes. No girdle sensation.

In the blood and spinal fluid the Wassermann test was negative: only 1-2 cells per c.mm. were present in the latter.

Mentally she was slow and heavy. There were no psychotic symptoms, but she had a host of neurotic complaints not necessary to detail here.

A diagnosis of hypothyroidism was made and she improved progressively under thyroid and Lugol's solution. At times she could not afford to buy the 2–5 grains of thyroid ordered and she would relapse. The special point to be emphasized was that both the pupillary and tendon reflexes improved very definitely, to relapse with the general mental state as she became careless in medication. She was seen fairly consecutively once a month for two and a half years, and then a few times in 1919 and once in 1920, since which time no word has been received although follow-up letters have been written. Every time she took her thyroid regularly she improved particularly as to the mental state and as to her neuritic condition. She had one of the 'gastric' attacks in six years observed.

Case II.—Mrs. Y., married, aged 36 in 1917 when seen as a patient. I had known this young woman for some years. She was a friend from childhood of an assistant of mine in the medical school. She was then about 20 or 21 years of age, a bright active alert young business woman, a highly competent secretary judged by the salary she earned and the steadiness of her employment in very responsible corporations and her intelligent industrious capacities.

I copy the history as reported in 1921 with slight addition from my notes.

She had a very complicated and full history from the time of her youth, which was made difficult by a religious fanatical father, followed by a seduction by a physician at sixteen. One sister died of tuberculosis and one sister was epileptic (completely negative luetic tests of this sister at Craig Colony, N.Y.). She then went on the stage, as a chorus girl, where she remained for two years. She then had a Neisserian infection, later, at twenty, severe scarlet fever with arthritis, and later a salpingitis required a complete hysterectomy in 1907. She was very intelligent and capable, she made good and had held for some years a very responsible position as private secretary with large corporate interests, and when I saw her, for the present difficulty, she was married, a woman of social position, means, accomplishment and charm, in spite of the adverse beginnings.

She had consulted her physician who, on reference to a local specialist, had told her she probably had a beginning of locomotor ataxia. She later came to New York on a motor trip which was hurried and only permitted a cursory examination.

The salient features of her early social history were as noted. In addition in 1900 she had had a 'big fever sore' on her lip which had lasted several

weeks. The doctor burned it and there was a hole for two to three weeks. There were no eruptions of any form following this. In 1904 she had an ulcer on the left thigh.

The present situation began about 1916. She had feelings of great uneasiness: shooting pains in the legs and arms; at times a band of anæsthesia unevenly distributed about the waist. Previously and about one or two years after the hysterectomy, she occasionally had severe attacks of projectile vomiting, lasting two days. At times she would have two of these in a year. The pain and cramps were terrific; she had not had one for over a year, but while in New York had a very severe attack of diarrhœa with cramps and pains which she said were a sort of incomplete or modified stomach attack. She was always cold and ' goosefleshy '; had frequent and severe colds; was nervous and fidgety; shook all over; was usually constipated, save when the periodic diarrhœas took place. In June, 1917, she had a circular zoster attack, with areas of paræsthesiæ of a ' creeping ' or ' crawling ' kind that persisted.

Examination at this time showed a well built woman with moderate adiposity. The skin was hard, cold and marble-like, very pale and anæmic. Goose fleshing was evident; the pilomotor reflexes irregularly hyperactive. Red dermographia was present and of moderate intensity. The eyebrows were very thin and the nails were fragile, the hair also. No sweating.

There was slight dulness in both apices. Whereas the skin in general was was cold and pallid and marble-like, she had the bright red cheeks of the English girl (she was born in Canada). She had some tachycardia at times although the pulse generally was slow. There were no other noteworthy general features. The lips were not markedly thickened nor the tongue swollen although the mouth was dry.

Neurological Examination: Patient had had since birth a slightly twisted nose and she said she had always felt she did not smell well. The eyesight was excellent. The fundus showed no changes. Pupils reacted slowly to cocaine when fundus was examined. The ocular movements were ample and free in all directions. Slight palpebral fissure irregularity. There was a definite inequality in the pupils. They were both under 2·5 mm., the right being the smaller by 1 mm. at least. The direct light reaction was very sluggish; classically myotonic. In the dark room it was more active, and there was slight slowness of the convergence reaction which was less marked in the dark room. The sympathetic reflex was present. The consensual light reflexes were not elicited. She had never had diplopia. There was no nystagmus. Fifth and seventh nerve functions normal. Hearing was less in the right ear (scarlet fever?).

Upper Extremities: Beyond the dermographia there were no objective findings in the upper extremities. There were no ataxias; no tender nerve-trunks; no appreciable losses in tendon-reflexes.

Trunk: No objective findings. No anæsthesia, or girdle sensation. Marked red dermographia persistent.

Lower extremities: No paralyses. Legs felt heavy. There was a very slight swaying when standing on one leg. Both knee-jerks were diminished and achilles-reflex absent. There was no ataxia on knee-heel tests. No loss of bone conduction. Position sense normal. Cold extremities. Sensory discrimination of all qualities normal.

Laboratory findings: Blood: 2 per cent. eosinophils. Blood pressure 155/105. Negative blood and spinal fluid Wassermann test; 7 cells per c.mm. Faint trace of globulin. No sugar. There were two later repetitions of the laboratory tests at six months' intervals (one at St. Luke's Hospital, New York); all

tests negative: 2 cells and 3 cells, respectively. There had been, previous to my seeing her, one reported plus blood Wassermann from a local laboratory.

A course of one hundred one-grain tablets of desiccated thyroid with cooked thyroid once or twice a week was advised. The local neurologist was written to that a dysthyroid syndrome was possible (after hysterectomy, overcompensatory gonadal thyroid activity with thyroid exhaustion) (she had had some slight swelling of the thyroid after the hysterectomy), and a careful revision of the material was suggested, since she showed unmistakable dysthyroid vagotonic stigmata.

The war activities withdrew all medical supervision and as the patient was a long way off in Western Canada it was difficult to trace further the situation from a technical point of view. She grew better, intermittently. At one time in 1919, following a continuous use of thyroid, the puffiness in the neck returned, her breathing was affected, headaches increased; she became jumpy. These symptoms disappeared after discontinuance of the drug.

The last reports in 1920 were that she had had no gastric attacks, no diarrhœas, no constipation. The arthritis was better, the pains were gone, her fatigue was better, though she had not the endurance of former days. Long letters indicated also definite unconscious psychogenic factors of difficult adjustment with reference to frigidity.

DISCUSSION.

There is little need for extended discussion. Concerning the signs of minor hypothyroid disturbance, Levi and Rothchild have summarized them with great precision and for the main it is well to recognize that scanty hair distribution, frilosity, arthritic, neuritic, and trophic disturbances may arise on the basis of dysthyroid function. The patients illustrate that there is a certain trend in a submyxœdema which may be confused with a tabetic syndrome.

One or two points may be touched upon which are of less well recognized significance, and as certain of these features run across the typical tabetic trail they may be commented upon, leaving for a future time a more extended and elaborate discussion.

It would appear that the dysthyroid condition can bring about a typical vagotonic state which can give rise to the characteristic crises of a similar vagotonic state which lies behind the crises of tabes. Eppinger has already suggested the explanation of the crises of tabes on a vagotonic hypothesis. Hence these ideas may be extended in two directions. The one has already been suggested; the other is that in tabetic pathology the fact of vegetative sympathetic pathways becoming more seriously involved in certain points permits the vagotonic reaction; and that it is possible that concomitant dysthyroid pathology is a factor in the constellation of the constitutional pathology to aid in the production of crises of many kinds. Hence it may be read that the syphilitic infiltration process in the incoming vegetative fibre-pathways, plus the dysthyroid modifications of the effector pathways, plus other factors (I myself have insisted that psychogenic factors may be added

in the constellation, in some instances bringing about the dysthyroid function) results in the crippling crises of tabes.

I am also disposed to analyze the tabetic arthropathies along similar lines of a ' constellation pathology.'

The pupillary reflex pathway modifications are probably much more complex, since the phyletic complications of the evolution of the light receptor mechanism of the eye are as yet far beyond our ability to unite into a satisfactory generalization. The vegetative reflex arcs of the intestinal and bone integrations, though still far from being completely resolved, yet are probably much simpler in their phyletic history and hence nearer being satisfactorily explained by our present day intellectual symbols.

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Editorial.

THE CLINICAL METHOD IN NEUROLOGY AND PSYCHIATRY.

ALTHOUGH the clinical method has to its credit innumerable advances in the twin sciences which concern us, there now appears to be a general feeling—tacit, implied, or openly expressed—that it has seen its best days and has no more worlds to conquer. The wave of clinical research, which began towards the latter end of last century, is declared to have more or less spent itself. Work of pioneers in neurology, such as Hughlings Jackson, Gowers, Charcot, or Oppenheim, of Kraepelin and others in psychiatry, was based largely if not solely on the bedside method, on painstaking clinical observation, and on deductions and inferences therefrom; it was substantiated—to some extent—by acquisitions in pathology, but in essence depended on study of symptoms and led to generalisations which in turn paved the way for still further accretions to knowledge. Now, we are assured, the procedure is becoming sterile. The neurologist, we are told in the recently issued Report of the Medical Research Council, must wait on the chemist and the physiologist; and perhaps the psychiatrist, by analogy, on the psychologist—if not, indeed, on other experts as well. No doubt the immense output of work on the clinical side, which helps to fill the pages of journals, reviews, quarterlies and annuals the world over, cannot in the nature of things go on for ever; and possibly we should no longer expect any strikingly new contribution such as those which in former times so impressed the medical readers of the day as to result in the naming of diseases after their discoverers. The truth is, however, that that epoch is not yet finished with; have not comparatively recent years witnessed the advent of 'Swift's disease,' 'Economo's disease,' 'Jakob's disease,' and others that might be mentioned? Whether such nomenclature is desirable or needful is beside the point; for the significance of the facts is, that by clinical investigation the sum of neurological knowledge has been added to. It would be unwise to insinuate that these possibilities are at a terminus.

Another feature of the clinical side of study is, that certain diseases come and go with some periodicity, or at irregular intervals, dying down seemingly only to appear again—either in the same or in some modified form. Such recrudescences are, quite properly, accompanied by a spate of articles in current medical literature, representing fresh opportunities for research and without question again adding their quota to knowledge. For example, the epidemics of cerebrospinal meningitis during the War might be quoted, or repeated epidemics of anterior poliomyelitis, or of encephalitis; during these visitations it must in all fairness be admitted that the clinician has often been able to accomplish much, not merely by observation but also by treatment. He cannot, of course, dispense with the aid received from the pathologist, biochemist, or bacteriologist, but it is he who from his position can direct lines of study and can apply what the others have been doing; they, on their part, depend on the clinician to a considerable degree for material.

To decry the rôle enacted by the clinician therefore becomes unwarranted; and it is equally improper to depreciate the scope of his labours. He has frequently been in advance of the pure scientist when it comes to recognition of novel phenomena, even though he may be well aware of his limitations. For instance, the peculiar manifestations of narcolepsy and cataplexy have of late engaged much attention; aside from their intrinsic interest, they are undoubtedly calculated to throw light on cerebral function—they have already done so; but as yet such data are beyond the ken of the experimentalist, who, if he takes the matter up in the laboratory, must clearly recognise how he is indebted to the clinician for the original discovery. The phenomena of catatonia, again, have been familiar to the bedside observer for generations; and though now the experimentalist is reproducing them in some degree by resort to an alkaloid such as bulbocapnine his work takes its bearings from that of his predecessor in the field.

From another point of view, it has been not unusual to find that the labours of the morbid anatomist and microscopist are rather minimised, in the sense that symptoms and lesions cannot be correlated by any rule of thumb, that end-results do not explain dynamic processes, and that causative factors differing widely in themselves are capable of inducing the same series of changes in the nervous system. True though these statements are, and however justifiable the allegation that the pathologist

is compelled largely to ignore the factor of time and tempted to draw inferences from a static picture that can hardly apply to a living process, nevertheless he has made, and will continue to make, notable contributions to knowledge without which many a disease would be more obscure even than it is. For example, the curious group of conditions classed as cerebral sclerosis cannot be understood except by reference to their underlying pathology—proof of which averment is furnished by Dr. J. G. Greenfield in the present number of this JOURNAL.

But despite arguments of the kind, which might be readily supplemented by others not now sketched, candour compels the admission that neither neurologist nor psychiatrist can hope to prosecute his work to its solution without the assistance of others not of the fraternity. For years the affection known as subacute combined degeneration was known mainly if not solely to the former; the general physician took little cognisance of it, still less the laboratory worker. Now, the neurologist as such does not perhaps occupy the same foremost place in connexion with it; or, rather, he sees his limitations, for its clinical and pathological syndromes are fully established, yet neither of these has provided the clue to its pathogenesis. Should it in reality turn out to be allied to deficiency diseases the biochemist has the honour of leading the van. Similarly, in respect of dementia præcox, if recent experimentation (clinical, it is true, but not psychiatric in the narrow sense) proves dependable, that 'disease' must be approached not perhaps any less from the standpoint of a disordered psyche but more from that of somatic disturbance.

Meanwhile the neurophysiologist, who perhaps in the seclusion of his laboratory hears echoes of clinical controversies, claims, assertions and speculations, probes ever deeper into the arcana that underly all nervous activity; he may know that the word 'inhibition' comes readily to the lips of the clinician, but he is seeking not a verbal but a physiological explanation of the process. He has more knowledge than the clinician always recognises of the intimate nature of the nerve-impulse, the differences between fibre and cell responses, of excitation and inhibition, of reflex action, and it is merely a question of time when—as has happened before—the latter more or less suddenly becomes alive to the fact that the other has solved some of his problems for him.

Evidently, then, it behoves the neurologist and psychiatrist to widen, not to restrict, their purview; they must not only welcome the aid given by other workers but must be on the alert to see its applicability to the subjects that interest and occupy them. Their corner of the vineyard is not sacrosanct; and if its confines are being invaded by labourers from elsewhere the advantage is mutual. No good reason exists for believing that the clinician must stand aside in consequence; on the contrary, his chances for further observation are thereby increased.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[145] **The brain problem in relation to weight and form.**—Henry H. Donaldson. *Amer. Jour. Psychiat.*, 1932, xii, 197.

As a result of this study, no method of assorting brains on the basis of weight, proportions of parts or the weight of proportional areas of the cerebral cortex in the several lobes, serves to classify them according to intellectual capacity. If, however, the subject is attacked from the side of the nutrition of the brain, it appears that the vascular tree, represented by the vessels of the pia, is better developed in the intellectuals. This apparently means a better blood-supply to the cortex, and it is unquestioned that such would contribute to a better performance. Finally, variations in the composition of the blood serve to modify the activities of the neurones, and though the evidence is at present from the field of pathology, it is a fair inference that for individuals called ' normal,' blood composition may be a significant factor in determining the grade of mental processes.

C. S. R.

[146] **The normal histology of the intradural filum terminale.**—John Watson Harmeier. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 308.

The filum terminale, the slender glistening thread which is prolonged downward within the vertebral canal from the end of the conus medullaris, finally anchoring the spinal medulla to the back of the first piece of the coccyx, is described as being about 6 inches (15 cm.) long. Down to the level of the second sacral vertebra it is enclosed with the surrounding nerve roots within the dura mater. This portion is called the filum terminale internum, in contrast to the portion distal to it to which the dura mater is directly applied, and which is called the filum terminale externum.

A histological study of material collected from 34 consecutive cases showed that the filum terminale internum contains all of the elements found in the spinal cord; a large vein and two small arteries are found in association with the neurogenic tissue; the more distal the segment examined, the less

neurogenic tissue is found and the greater is the decrease in the component parts of the neurogenic tissue. Tumours of the filum terminale internum should be similar in type to those found in the spinal cord.

R. M. S.

[147] **On a constant motor phenomenon of normal sleep—physiological hypnic myoclonus** (Su di un fenomeno motorio costante del sonno normale: le mioclonie ipniche fisiologiche).—L. DE LISI. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 481.

SLIGHT muscular movements are normal in man and animals in the first half hour of sleep. They are commoner in the first sleep and in light sleep. They are as a rule small in extension, unorganized and asynergic, but sometimes amount almost to a general convulsion. They may occur in any part of the musculature and are not influenced by external stimuli or change of position.

No definite explanation of the phenomenon is given.

R. G. G.

NEUROPATHOLOGY.

[148] **Measurement of the muscular tone in man during the action of bulbocapnine injected intravenously** (Misurazioni del tono muscolare nell'uomo durante l'azione della bulbocapnina iniettata per via endovenosa).—U. DE GIACOMO. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 110.

IN ten experiments performed by the methods of Marigold and Spiegel the behaviour of the resting tone of muscle was studied in its dual aspect of duration and resistance to stretching during the period of action of ·1 grm. of bulbocapnine injected intravenously.

In harmony with the results of previous experiments bulbocapnine caused in nine experiments a first phase of relaxation and in five experiments a second phase of catalepsy. The subjects used were four mental defectives, two paretics, and two dementia præcox cases.

In the relaxation phase there was an abrupt lowering of tone followed by a return to normal. For many reasons this did not appear to be a mere relaxation of muscle but a hypotonia due to the direct effect of the bulbocapnine on the central nervous system.

In the cataleptic stage there is no increase of muscular tone; the condition would seem to be due to a ' psychomotor stupor ' and transitory interference with subcortical mechanisms.

R. G. G.

[149] **Action of scopolamine and carbon dioxide on catalepsy produced by bulbocapnine.**—A. S. Paterson and C. P. Richter. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 231.

One of the most characteristic features in the behaviour of monkeys under the influence of bulbocapnine is their tendency to hold on to any object placed within their grasp. A normal monkey with three limbs tied will cling to a horizontal bar brought within reach of the free hand but will drop from it and attempt to escape as soon as he is left hanging by the experimenter. A monkey similarly bound but given an injection of bulbocapnine will not only grasp the bar but will hang from it for several minutes. This hanging response, probably closely related to the grasp reflex of the new-born infant, is doubtless a part of the catalepsy produced by the drug, in that the animal continues to grasp the bar once it has obtained a hold, just as it tends to maintain any other posture in which it is placed.

The strength of the reflex can be determined by measuring how long the animal will hang from a horizontal bar. By testing the animal at regular intervals after the injection of bulbocapnine, a curve can be obtained of the effect and its intensity at various times following the injection.

With these curves as a basis, a study was made of the effect of carbon dioxide and scopolamine on catalepsy produced by bulbocapnine.

Scopolamine caused a marked prolongation of the effects of bulbocapnine from 60 or 80 minutes to 300 minutes or more, despite the high degree of muscular relaxation present. The animals showed no sign of recovery from the stupor.

Carbon dioxide has different effects, depending on the dosage. In lower concentrations, from 15 to 25 per cent., administered for from one to two minutes, it caused an increase in the length of time that the animal hung immediately after administration of the gas was discontinued. In higher concentrations, from 35 to 50 per cent., with the appearance of anæsthesia, it caused a decrease in the hanging response with a subsequent increase, the total duration of the effect of bulbocapnine often being increased, as it was with scopolamine.

In marked contrast to scopolamine, however, carbon dioxide given in a higher concentration caused a transient disappearance of the hanging response for a few minutes following the administration of the gas, and a complete respite from the stuporous symptoms. The effect was practically identical with that produced by the gas on catatonic symptoms, in man.

The fact that carbon dioxide and scopolamine produced an increase in the hanging time suggested that these agents have a positive action on the response and that they might produce it when given without bulbocapnine. It was found that both carbon dioxide and scopolamine, when given alone, elicit the grasp reflex and the hanging response.

To this extent they are not counteracting agents. However, it is pointed out that carbon dioxide might have either an augmenting or a counteracting effect, depending on the concentration of the gas.

On the basis of these observations, the relationship of catalepsy produced by bulbocapnine to human catalepsy and paralysis agitans is discussed.

R. M. S.

[150] **Effect of rœntgen rays on the central nervous system.**—R. S. Lyman, P. S. Kupalov, and W. Scholz. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 56.

Four adult dogs received heavy dosage of rœntgen rays on the occipital part of their heads. Physiological variations followed irradiation, as determined by conditioned reflexes. Histological examination showed diffusely scattered degenerative changes in the precapillaries and capillaries of the brain six weeks after irradiation. One dog was allowed to live for six months after irradiation and in the fifth month there developed signs of diffuse damage to the functions of the cortex and subcortex. At autopsy the changes in the precapillaries, capillaries and larger vessels of the brain had progressed and there were marked hyaline degeneration and obliterating sclerosis of arterioles with resulting areas of complete and incomplete necrosis.

The conclusion is reached that rœntgen rays may alter the function of the central nervous system, but their action does not seem to be primarily on the cells of the cerebral cortex. After a high dosage in adult dogs there may be some lowering of excitability of cortical cells, as determined by conditioned reflexes, but this appears to be secondary to changes in blood supply. Even when directed at a single area of the brain, irradiation gives a diffuse physiological and pathological effect, involving the subcortex as well as the cortex, and probably the vegetative nervous system as well. Accordingly, in spite of some photographic evidence that the physical action of the rœntgen rays can be fairly localized inside the skull, there is little hope of selecting one spot in the brain for cross-fire with a small beam and of increasing the vascular permeability at that spot by irradiation, and then treating through the blood-stream so that the desired area will get relatively more medication than other parts of the brain.

R. M. S.

[151] **Meningeal permeability and the socalled hæmato-encephalic barrier** (Contributo allo studio della permeabilita meningea e della cosidetta barriera ematoencefalica).—O. Rossi. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 92.

It was found that malarial parasites could be introduced into the cerebrospinal fluid and cross the barrier into the blood even in patients in whom there were no evident lesions of the structures composing this barrier. The

malarial infection provoked by this method has the ordinary characters of that transmitted from man to man. It is not possible so far to establish the indications for producing malaria in paretics by the lumbar route. The introduction of malarial blood into the dural sac does not seem to be attended by harmful results.

R. G. G.

[152] **Gliomas arising from the region of the cauda equina.**—James W. Kernohan, Henry W. Woltman, and Alfred W. Adson. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 287.

The histological structure of the spinal cord is similar to that of the brain, and in their study of intramedullary tumours the authors found them to correspond in type with tumours arising from the substance of the brain. There was considerable difference in the distribution of the numbers of tumours in the various types of glioma, and there was a slight variation in the characteristics of the individual tumours in some of the subgroups, but for the most part they corresponded closely. Study of the normal conus medullaris and filum terminale and the observations of Harmier have shown that they contain all the elements that are present in the spinal cord. There were relatively more ependymal cells, sometimes growing as tubules but usually without any definite cell arrangement except in the ventriculus terminalis, than any other type of cell, and consequently it should be expected that ependymomas would be relatively more common than in the spinal cord. This proved to be true, as 68 per cent. of tumours arising from the region of the cauda equina were ependymomas.

R. M. S.

[153] **Nerve degeneration in poliomyelitis.**—Herman Chor. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 344.

The motor nerve-endings of Macacus rhesus monkeys have structural characteristics common to other mammals, with slight variations in finer details, such as an elaborate terminal branching and abundance of neurofibrillar end-loops and buds.

Following nerve section, degenerative changes occur which are evident by the end of twenty-four hours. These are ushered in by irregular swelling and staining of the fine terminal twigs of the neurofibrillar end-brush. By the second day, fragmentation of the neurofibrils appears, and the axon and myelin sheaths outside the muscle fibre show irregularities in contour and in staining qualities. After the third day, the ending is replaced by a granular strand which goes on to complete absorption, and the more proximal portions of the motor nerves show advanced fragmentation of axons and segmentation of myelin. Subsequently, only remnants of nerve-endings can be made out, although occasional persisting neurofibrils are encountered.

In poliomyelitis in the preparalytic stage the motor nerve-endings appear normal, but with the onset of paralysis perceptible changes occur. These changes, associated with the appearance of muscle weakness and advancing through the various stages of paralysis, are identical in most respects with those observed after section of the nerves. The distinguishing feature, however, is the lack of uniformity in the degree of damage displayed by the endings at any given time in contrast to the uniformity found following section. This may perhaps be explained by the unequal involvement of the anterior horn cells in poliomyelitis and also by the plurisegmental innervation characteristic of striated muscle.

Direct contact of motor nerve-endings with the virus of poliomyelitis (as implied by saturation of muscles with the virus) failed to produce perceptible histological changes sufficient to indicate primary involvement.

These findings lead to the conclusion that the changes in the motor endings observed in experimental poliomyelitis are secondary to primary damage of the anterior horn cells.

R. M. S.

SENSORIMOTOR NEUROLOGY.

[154] **Three cases of cerebral cysticercus diagnosed during life** (Considerazioni su tre casi di cisticercosi cerebrale diagnosticati in vita).—C. Rizzo. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 503.

The present case and two others previously described in the *Rivista* are reviewed and considered. A full description of the morbid anatomy with illustrations is given, and a clinical description of the course of the disease with the mental disturbance. Diagnosis in life rests on the presence of eosinophils in the cerebrospinal fluid; these may not be present in the blood if the cysticercus is confined to the central nervous system.

R. G. G.

[155] **Neurinomata: their association with acromegaly and possible malignancy** (Contributo allo studio dei neurinomi con particolare riguardo all'associazione con acromegalia ed alla loro trasformazione maligna).—C. Fittipaldi. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 521.

Two cases are described and the literature is reviewed. The first case presented no unusual symptoms, but the second showed a clinical syndrome of acromegaly and was evidently malignant, as proved by clinical and histological evidence. The author thinks that the acromegaly was due to the presence of a blastoma of the same type as the peripheral tumour in or about the hypophysis. The malignancy of the tumour showed itself in the rapid growth of the primary neurinoma and by the presence of metastases.

To this clinical course a histological malignancy corresponded, characterized by a notable modification of the neurinomatous cells in the presence of karyokinesis and the infiltration of the vessels. The author however, in addition to suggesting the existence of malignant neurinomata like the observed case, thinks that maltransformation of these blastomata can result also from anaplasia of the connective-tissue in the tumour.

R. G. G.

[156] **Circumscribed cerebral atrophy or Pick's disease.** (L'atrofia cerebrale circoscritta o malattia di Pick).—S. GULLOTTA. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 600.

THIS condition is described in detail with special reference to the localization of areas of degeneration. Involuntary movements are common, as are disturbances of speech and memory. Diagnosis from other forms of dementia is difficult. The prognosis is bad and treatment ineffective.

R. G. G.

[157] **Epilepsy and narcolepsy associated with hyperinsulinism.**—SEALE HARRIS. *Jour. Amer. Med. Assoc.*, 1933, c, 321.

THREE cases of epilepsy and one of narcolepsy are described, and it is suggested that there may be a possible relationship with hyperinsulinism. If there be a distinct type of epilepsy associated with this condition, it will probably be found in young adults and there is ample reason to believe that some such cases may be controlled by a low carbohydrate, high fat diet with frequent feedings, sufficient to maintain the blood sugar level at a point above which hypoglycæmic convulsions occur. A study of the blood sugar after the use of bromides in one case of epilepsy with an apparently fixed hyperinsulinism curve suggests that this drug may control the convulsions in this type by inhibiting the secretion of insulin, thus maintaining the blood sugar level above the point at which hypoglycæmic convulsions occur. Bromides are not advised here because of the harmful effects of bromism. Since a number of recurring convulsive attacks have been proved to be due to insulomas, it seems probable that in some cases of epilepsy associated with hyperinsulinism, adenomas of the pancreas may be found to be the cause of the convulsions, and such cases may be amenable to surgery. The excellent results of surgery for the relief of these convulsions presage operations becoming more frequent.

C. S. R.

[158] **On the hereditary transmission of idiopathic tremor** (Sulla trasmissione ereditaria del tremore essenziale).—G. PINTUS. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 1.

THE author studied the hereditary transmission of idiopathic tremor in a family of the province of Cagliari whose family tree comprised some 200

members. He came to the conclusion that a transmitted tremor was manifested in the family; it sometimes assumed the form of a finely vibrating tremor and sometimes of an intention-tremor with large, slow movements. The transmission was that of a dominant single gene and appeared when a dominant heterozygote crossed with a healthy recessive. The law of Mendelian dominance seemed to be exemplified almost exactly.

The family did not seem to be specially prolific or long-lived, but the author did not have at his disposal the statistics of the rest of the province. The diagrams of the family tree are given.

R. G. G.

[159] **The influence of flexion and extension of the head on the postural reflexes of the lower limbs** (L'influenza della flessione e dell'estensione del capo sui reflessi di postura degli arti inferiori).—S. GARETTO. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 167.

FLEXION and extension of the head determine a reinforcement of the postural tone of the lower limbs. This reinforcement is not due to a diffuse hypertonia determined ' from without ' but to a postural reaction of reflex origin induced by the change in the position of the head; that is to say, from proprioceptive stimuli upon the muscles of the neck. Flexion of the head causes an increase of tone of the muscles of the front of the leg and extension of the head an increase in those of the back of the leg.

R. G. G.

[160] **Subacute combined degeneration of the spinal cord.**—N. W. WINKELMAN and CHARLES DAVISON. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 317.

IN a study of 25 cases clinically and pathologically diagnosed as pernicious anæmia with changes in the spinal cord 20 presented nothing unusual in the histological pictures. These showed the typical funicular myelopathy that is characteristic of this disease.

In five cases there was, in addition, a perivascular round-cell infiltration, mainly in the posterior and lateral portions of the spinal cord.

In an effort to determine whether the condition was inflammatory or degenerative, differential staining methods were used. In the cases of pernicious anæmia, the majority of the perivascular cells proved to be of the compound granular cell type. In contrast to this, dementia paralytica was studied by the same methods, and the plasma-cell and lymphocyte were found to be the predominating round-cells. The term ' small round-cell infiltration ' is therefore a noncommittal description of cells lying in the perivascular spaces. An effort should be made to determine the type of cell in the exudate, because it gives a clue as to the nature of the pathological

process that is going on. While it is true that degeneration is part and parcel of an inflammatory process, inflammation is not usually part of a degenerative condition.

R. M. S.

[161] **Syphilitic amyotrophy.**—R. P. MACKAY and GEORGE W. HALL. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 241.

FOUR cases are reported as representing a form of progressive muscular atrophy due to syphilis.

As a group, these cases exhibit histological, physical, and serological evidences of syphilis; the atrophy often begins with pain, most frequently involves the upper extremities and not uncommonly occurs first in the shoulders and simultaneously or later in the hands, and may be very patchy in its distribution; the pyramidal tracts may or may not be involved.

A review of knowledge of the pathology of the group reveals much confusion, but seems to indicate a syphilitic meningoradiculomyelitis as the basis for most cases, with the question of a truly parenchymatous and selective syphilitic disease of the anterior horn cells as yet unsettled.

Antisyphilitic measures, vigorously and actively adopted, offer therapeutic hope in an otherwise hopeless neurological field.

R. M. S.

Psychopathology.

PSYCHOLOGY.

[162] **Effect of conscious wish upon dream content.**—LOWELL S. SELLING. *Jour. of Abnorm. Soc. Psychol.*, 1932, xxvii, 172.

THE author's findings may be thus summarized. When an individual indulges in continual conscious wish phantasy, even though it be non-sexual, it makes itself manifest in the dream. Although any crime may be based on a maternal, paternal, or other complex, the emotions are so stirred by punishment or other conditions resulting from incarceration, that the non-incarceration existence tends to blot out significant dream material concerning the crime. The paternal situation may often determine the lines upon which a dream will be formed, but there is no consistency between intelligence and dream content. A minimum of dreams was found carrying a content which could be interpreted along Freudian lines and in which a subconscious stream of thought was suggested. A simplification of dreams occurs with an increased simplicity of environment (prison environment). These findings tend to suggest that while there may be latent (Freudian) content in non-neurotic individuals, most dreams may be explained more simply than is usually the case.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[163] **Encephalitic and hysterical syndromes** (Sindromi encefalitiche e sindromi: isteriche patogenesi).—V. M. Buscaino. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 33.

The author has studied the literature of non-purulent encephalitis especially in relation to oculogyric crises and drawn from it the evidence which can best contribute to the study of the genesis of the hysterical syndromes. He comes to the general conclusion that the extracortical zone (basal ganglia and mesencephalon) is of major importance in the genesis of the most important elements in the hysterical syndrome.

These conclusions are at present general and must be worked out in greater detail later, with the documentation of the objective disturbances of the vegetative innervation of the hysteric—disturbances quite independent of psychic activity (especially in relation to the asymmetry of vegetative innervation between the two sides of the body). Attention must also be given to the documentation of hysterical disturbances of an emotional and vegetato-emotional character with special relation to the common site of emotional and vegetative activity in the basal ganglia and mesencephalon.

R. G. G.

PSYCHOSES.

[164] **A note on the relationship between the blood cholesterol and hyperglycæmic index in manic-depressive psychosis.**—Madeline R. Lockwood. *Jour. of Ment. Sci.*, 1932, lxxviii, 901.

The hyperglycæmic index and the blood cholesterol were examined in 24 cases of manic-depressive psychosis, and a relationship was shown to exist between the two values. The blood cholesterol content shows a delay in the return to normal as compared with the glucose tolerance curve. When the hyperglycæmic index falls to zero, the cholesterol level lies at the upper limit of normal. A rise in the index is associated with quantitative changes in the blood cholesterol of such a nature that the two values bear an inverse ratio to each other. Three cases in which the test has been repeated at different phases of the illness show that variations in the index are accompanied by variations in the cholesterol level. The possible relationship between these findings and a disordered endocrine balance is briefly discussed and an explanation suggested.

C. S. R.

[165] **Psychosis: its importance as a presenting symptom of brain tumour.**—Leo J. Adelstein and Martin G. Carter. *Amer. Jour. Psychiat.*, 1932, xii, 317.

The incidence of brain tumour as a clinical entity is approximately 1 per cent. Psychosis per se may be the first presenting symptom. All cases

presenting a psychosis should receive a thorough neurological examination to rule out a possible organic basis in the nature of an intracranial neoplasm. Visual and auditory hallucinations are of localizing value only if distinct and apart from a psychosis. Mental phenomena associated with brain tumour may be regarded as focal in nature only if the neurological examination will bear out the localization. In the series of cases here studied, the most marked mental changes were noted in those involving the frontal lobes and the corpus callosum, which coincides with the opinion of various authorities.

C. S. R.

[166] **A study of the sexual life in psychoses associated with childbirth.**—E. W. ANDERSON. *Jour. of Ment. Sci.*, 1933, lxxix, 137.

FIFTY cases of psychosis, occurring in association with childbirth, were studied with special reference to the sexual life, and compared with a like number of cases of psychosis in married women of approximately the same age-period where the puerperium was not the precipitating factor. No material differences were revealed in the prepsychotic sexual life of the two groups. Persistent frigidity cannot be reckoned a distinctive or even constant feature of the sexual constitution of the cases in the puerperal group. Masturbation and eroticism were only slightly commoner in the puerperal group, and cannot be regarded as characteristic or even particularly common features of the group. In the whole series of 100 cases masturbation only occurred in eight. The attitude to both husband and child in puerperal psychoses is inconstant, and aversion to either or both is by no means constantly or even very frequently found. The figures relating to previous attacks taken in conjunction with those on the prepsychotic sexual life, fail to support the view that the onset of a psychosis associated with childbirth is a product of a morbid sexual constitution, with the puerperium acting as a specific precipitating factor. The latter must be regarded in the same way as any other precipitating factor whatever.

C. S. R.

[167] **Use of carbon dioxide mixtures in stupors occurring in psychoses.**—F. C. D'ELSEAUX and H. C. SOLOMON. *Arch. of Neurol. and Psychiat.*, 1933, xxix, 213.

THE essential purpose of the authors has been to determine the cause or causes instrumental in arousing patients from their stuporous condition when they are given carbon dioxide to inhale. This arousal has been called the mental response, and on the evaluation of it depend the judgment as to the relative efficacy of the various procedures used and the judgment as to what are the factors causative in bringing it about.

One is forced to rely on the overt behaviour of the patient for making this estimation. This includes muscular movement of both the voluntary

and the involuntary sort, verbal utterances and such elusive yet real factors as the gleam in the eye and modulation of the voice. Furthermore, there are a number of attributes of each of these forms of activity which are used in estimating the mental activity of a person. Some are the degree of spontaneity of the act, or, on the other hand, the amount of stimulation needed to produce it; the relative complexity of the movement or verbal utterance and of the stimulus calling it out; the speed of initiation and of performance, and the completeness of performance; the purposiveness, adequacy and degree of relevancy of the act to the stimulus and to the total situation.

Besides the degree of normality, two other factors must be considered: (1) the amount of response obtained or, in other words, the increase in overt behaviour over that present in rest, and (2) the duration of the response, that is, the length of time during which the change in overt behaviour is present.

In addition to carbon dioxide, experiments were made with nitrous oxide and diathermy. The stimulating effect on catatonic patients of all these different methods indicated that no one or no combination of the observed physiological changes can be considered as essential to the obtaining of the response; the multiplicity of methods used with success hardly admits of a single and common physiological factor being responsible; the only known similarity between these procedures which may be responsible for the effect is some sort of shock to the body economy; yet other such shocks are not effective, nor are these always effective. There are various psychological factors which alter the response, at times increasing it or insuring it and at other times decreasing or even negating it. The various a priori hypotheses that have been advanced to explain the effect obtained, such as stimulation of the nerve structures by carbon dioxide or by a difference in the supply or utilization of oxygen, or a change in the metabolic and functional activity of the nerve cells as a result of changes in acidity, lack factual evidence for their support; they are neither proved nor disproved, but it can be said definitely that none of them is adequate to explain the phenomena in question.

R. M. S.

[168] **A clinical study of psychoses associated with various types of endocrinopathy.**—J. NOTKIN. *Amer. Jour. Psychiat.*, 1932, xii, 331.

THIS study indicates that the occurrence of psychoses in association with endocrinopathies of gross character is rather infrequent, inasmuch as only eight cases were found in a hospital population of 6,000 patients. The presence of three hypophrenics in this group reduces this number to a greater extent, as the endocrinopathies in mental deficiency are not infrequent. This infrequency seems to indicate that the relationship of the

endocrine dysfunction to mental disorders has been overrated considerably, especially if we bear in mind the frequent occurrence of endocrine dysfunction without psychoses. The stress which has been put upon certain disturbances in calcium and lipoid metabolism (cholesterol, lecithin) and offered as a possible etological factor in mental disorders does not seem to be justified, as the deviations for calcium and lipins in such overt endocrinopathies as are presented in this study are minimal. The lack of any definite findings on which an organic etiology can be built up does not entail having recourse to psychoanalytic doctrine. It seems, however, reasonable to assume that in a number of these cases the physical handicap is tied up with the dynamics of the mental trend.

C. S. R.

PSYCHOPATHOLOGY.

[169] **Crime and the endocrine glands.**—Louis Berman. *Amer. Jour. Psychiat.*, 1932, xii, 215.

This writer regards crime as due, in a ' Gestalt ' sense, to a perversion of the instinctive drives dependent upon a deficiency and imbalance of the endocrine glands. Certain types of crimes are associated with certain types of endocrine malfunctioning. Most criminals are derived from juvenile delinquents and most juvenile delinquents tend to become criminals. Endocrine imbalance and deficiency have been found to occur in about the same frequency and of about the same type in juvenile delinquents as in criminals. Endocrine treatment of the specific endocrine condition in juvenile delinquents has resulted in a correction of the delinquent behaviour. Juvenile delinquency and its sequel, crime, can be prevented by proper attention to the status of the different endocrines which contribute to the development of the normal social personality during childhood and adolescence.

C. S. R.

[170] **Mental disorders in siblings.**—Doncaster George Humm. *Amer. Jour. Psychiat.*, 1932, xii, 239.

This study was undertaken in the hope of gaining a better understanding of the causation of persistent criminalism, manic-depressive psychosis, dementia præcox, epilepsy, and mental deficiency. A particular object was to determine the relative importance of hereditary and environmental factors in that causation. The material especially gathered for this study consisted of 858 siblings of patients suffering from these disorders. Access was also secured to 214 pairs of twins, one or both of each pair being affected by one of these disorders. Of these 138 were dizygotic and 76 were classified as probably monozygotic.

It was found that the closer the degree of genetic relationship to an affected subject, the greater is the tendency to mental disorder. In monozygotic twins the incidence was as high as 93·5 per cent., with 69·8 per cent. in state institutions; in siblings as high as 17·8 per cent.; while in the unselected population 0·565 per cent. only were in state institutions. This would seem to justify the conclusion that genetic factors play a prominent part in the causation of the disorders studied. Of 74 pairs of opposite-sex twins, both were affected in 18 cases (24·3 per cent.); of 401 siblings of the sex opposite to that of their respective propositi, 59 or 17·1 per cent. were affected. Differences in age, great or small, between siblings and propositi had no appreciable effect. When comparing the occurrence of dissimilar disorders within the families studied, many siblings were found affected with disorders dissimilar to their respective propositi. There were several families in which there was coexistence of manic-depressive psychosis and dementia præcox; also even a larger number of families in which there was coexistence of epilepsy and mental deficiency.

A frequent familial coexistence of epilepsy with migrainous headaches, enuresis, and outbursts of rage was found. In a similar way there was an association between manic-depressive psychosis and cases of cycloid personality. A comparison was also made of the incidence of mental disorders in the two sexes, and in siblings of the same sex as their respective propositi as contrasted with that in siblings of the opposite sex. Of the 858 subjects investigated, 414 were males and 444 females. Of these 75 of the males (20·4 per cent.) and 62 of the females (15·2 per cent.) were afflicted with a mental disorder. In other words, incidence is, on the whole, greater in males.

C. S. R.

[171] **Some neuropsychiatric problems of the flight surgeon.**—Eugen G. Reinartz. *Jour. of Aviation Med.*, 1932, iii, 137.

When a cadet is referred for neuropsychiatric examination the attempt is made to put him at his ease and to get the greatest degree of 'transference' possible. He is permitted to tell his story without much suggestion or prompting. A certain something has made itself manifest on his mental horizon that bodes ill for him, the exact nature of which he is unable to fathom. The cause of the cadet's shortcomings is usually found to be an expression of the basic instinct of fear, clothed with symbolisms, repressions and rationalisations. Projection is not infrequently used. The cadet fastens his difficulties in flying on to something external. During the last training class of some 303 men, there were 17, who, because of potentialities, were brought to the attention of the Neuropsychiatric Department. Not all who had difficulty with flying came to that Department, since of the 303, starting

training, 50 per cent. were, for various reasons, excluded. Of the 17 men who were examined and who were on the edge of discharge from training because of the difficulties they were experiencing, 11 (64·7 per cent.) were adjusted to a greater or less extent. There is often projection on to the instructor who is ' not understood ' by the cadet or who ' does not understand ' the individual who is being taught. If the cadet is of an extroverted personality type the fear manifestation will probably be of a hysterical nature; if he is of the introverted type, his reactions will probably be of an anxiety nature. The individual having difficulty with his flying will usually be found to have some psychoneurotic taint. The so-called ' gastropath ' furnishes the largest number of the hysterical. They report stomach disorders, bowel disorders, indefinite, intangible pains, vertigo with or without nausea, headaches, heart disturbances and a host of other symptom—anything and everything, to remove them from a most painful situation of stress. The degree with which the cadet responds to treatment is indicative, to a certain extent, of the course he will pursue in his subsequent flying. The fact that a cadet has been found psychologically unfit for flying need not be held against him and the examiner may have the opportunity of helping the disqualified individual to adjust himself on a higher, better level, as he returns to his civilian pursuits.

C. S. R.

[172] **Amnesia in relation to crime.**—J. S. Hopwood and H. K. Snell. *Jour. of Ment. Sci.*, 1933, lxxix, 27.

The differentiation of true and simulated amnesia may be simple or very difficult. Amnesia cannot be diagnosed on the patient's word alone. A history of chronic alcoholism or of other indications of a psychopathic personality, including a previous loss of memory, is frequently elicited. A neuropathic heredity is to be expected in a fair number of cases. The character of the alleged amnesic period is of foremost value. In genuine cases the beginning and more especially the end are frequently blurred, and usually the loss of memory prior to the committal of the crime is of much shorter duration than that of the subsequent period before normal consciousness resumes control. When the amnesia is partial and the patient has a confused recollection of his acts, the ideas accompanied by marked emotion are remembered, while those with less emotional tone are not. Statements made soon after the crime showing a knowledge of the act are not incompatible with genuine amnesia due to a later repression. The crime is frequently without motive, and is carried out in a way which shows that the individual was not acting with premeditation, and he makes no attempt to escape from the consequence of his actions. A sudden return of memory almost certainly indicates malingering.

C. S. R.

[173] **Mental deficiency.**—Richard J. A. Berry. *Bristol Med.-Chir. Jour.*, 1932, xlii, 177.

One hundred and sixty adult feeble-minded women were examined with a view to ascertaining, if possible, the causative factors of the abnormal condition. This and other allied investigations into the subject seem to suggest, if not indeed to prove, that mental deficiency is a manifestation of improper development and not of disease, so that the problem becomes one of preventive medicine, eugenics and embryology rather than of curative medicine.

C. S. R.

[174] **Possible detrimental effects of experimental hypnosis.**—Milton H. Erickson. *Jour. of Abnorm. Soc. Psychol.*, 1932, xxvii, 321.

Literature offers little credible information concerning possible detrimental effects of experimental hypnosis, although replete with dogmatic and opinionated denunciations founded on outworn and untenable concepts of the phenomenon. Theoretical possibilities of detrimental effects are possible development of hypersuggestibility, the alteration of personality, weakening of the subject's perceptual powers in regard to reality and unreality, and lastly, the development of unhealthy mental attitudes and escape mechanisms. The literature is barren of controlled investigation of these problems. The author's own experience, based upon several thousand trances on approximately three hundred individual subjects, some of whom were hypnotized at least five hundred times each over a period of four to six years, reveals no evidence of such harmful effects. This clinical finding is further substantiated by the well-known difficulties encountered in deliberate therapeutic attempts to occasion desired thoughts in the personality. Accordingly, marked changes from experimental hypnosis appear questionable.

C. S. R.

[175] **A psychological case-study of the amyostatic-akinetic form of encephalitis lethargica.**—K. H. R. Edwards. *Brit. Jour. Med. Psychol.*, 1932, xii, 315.

We here have a history of a case of encephalitis lethargica in an individual who previously revealed an inhibited restrictive personality, possessed Kretschmer's asthenic constitution and a schizoid temperament. Infection probably occurred during an excessive fatigue period and the usual symptoms of the amyostatic-akinetic form of the disease became evident. There was an early appearance of depression and later a protracted convalescence and continued psychic torpor. These are explained by the former

existence of psychotic trends which were precipitated by the disease-process. During the course of the disease, instinctive mechanisms were, in general, stimulated or inhibited; some perversions appeared. Emotional responses became richer and more numerous. Character weaknesses were all emphasized and the psychopathic elements of his temperament became unusually prominent. Pseudo-neurasthenic states were noted in the protracted convalescent stage. It is suggested that infection in a more resistant system usually leads to the hyperkinetic form of encephalitis and in less resistant systems to the amyostatic-akinetic form. Severe infection would produce the somnolent ophthalmoplegic form in both more or less resistant systems. The danger of assuming a direct simple relation between manifest disorders and pathological lesions is stated. It is held that the psychological accompaniments of lesions interact among themselves and with pre-existing psychological experiences of the patient. That certain mental states are accompanied by specific anatomical lesions does not permit of a direct causal link between them. The view that in encephalitis there is an alteration of the thymo-psyche, leaving the noö-psyche intact, is criticised. It is suggested that intellectual functions are also involved even though this may not be observable. Pavlov's experimental work is used to explain the incidence and continuance of the somnolent and stuporose states. Both correspond to conditions in which severe loss of nearly all conditioned reflexes occur.

C. S. R.

PROGNOSIS AND TREATMENT.

[176] **Sulphur treatment in mental diseases : an experimental study of 100 cases.**—J. E. Dhunjibhoy. *Indian Med. Gaz.*, 1932, lxvii, 612.

From his investigations at the Rauchi Indian Mental Hospital the writer concludes that there can be no doubt that injections of sulphur in olive oil invariably produce pyrexia, the average temperature obtained being 102·1° F. Pain at the site of injection in every case was unavoidable but certainly bearable. These injections should be given unhesitatingly because they are perfectly safe and can be used for young and old alike. Earlier cases respond better to treatment than the old chronic cases and the earlier the treatment is instituted the greater is the chance of recovery. Sulphur merits a trial in all early cases of psychoneurosis and psychosis and seems definitely useful in chronic psychotics who have hitherto failed to respond to other methods of treatment. It also appears to be very useful in cases of benign and secondary stupors. Out of the 100 cases, 39 improved, 13 completely recovered, and no improvement of any kind was noticed in 48 cases. Of the

39 improved, 18 have since relapsed and the remaining 21 have still maintained the improvement. There were 37 manic-depressives in the group, and of these, 16 improved, six recovered, and 15 remained stationary. Out of 44 schizophrenics, 20 improved, four recovered, and 20 showed no change.

C. S. R.

[177] **Nembutal in mental hospital practice.**—DUNCAN MACMILLAN. *Jour. of Ment. Sci.*, 1932, lxxviii, 892.

IN this investigation the action of the drug by the intravenous route was tested on a series of 50 mental patients. It was concluded that nembutal is a rapidly-acting potent sedative. It assists in the investigation and assessment of the mental state of inaccessible and reticent patients. It has produced a curative action in early psychotics. In melancholia it exerts a prolonged calming action on agitation, and repeated injections have a beneficial effect. In cases of emergency, the necessity for a general anæsthetic may be obviated by its use. For these reasons, and also because of its sphere of action as a basal narcotic, it is a useful addition to the therapeutic equipment of a mental hospital.

C. S. R.

[178] **The treatment of the voluntary boarder.**—HENRY L. WILSON. *Jour. of Ment. Sci.*, 1933, lxxix, 102.

BETWEEN January, 1891, and December, 1930, 1,095 voluntary boarders were admitted to The Retreat, York. The results have been that 40 per cent recovered, 40 per cent. were relieved, and 20 per cent. were unimproved. In 1,170 cases under certificate, the results were 42 per cent. recovered, 31 per cent. relieved, and 27 per cent. unimproved. The conditions under which voluntary patients are received allow a number to leave against advice. Of the unrecovered boarders 44 per cent. left in this way. Under conditions of legal detention this number would be likely to be much less, and an approximate figure for the results then is computed to be:—recovered 53 per cent., relieved 30 per cent., unimproved 17 per cent. When their behaviour no longer confirmed their volition 170 cases were certified. The causes for this step were confusion, stupor, excitement, indecision, querulousness, suicidal attempts. Of these cases 51 per cent. ultimately recovered. Neither senile nor paranoid cases are at all times unsuitable for voluntary admission. The ease with which a voluntary boarder can leave is especially dangerous for potential suicides. Voluntary reception has enabled alcoholics and psychoneurotics to seek hospital treatment. Of the 1,095 admissions, 52 were of alcoholics, and 99 were of psychoneurotics.

C. S. R.

Reviews and Notices of Books.

Poliomyelitis. A Survey by various Writers, under the auspices of the International Committee for the Study of Infantile Paralysis. Baltimore: Williams and Wilkins Co.; London: Baillière, Tindall and Cox. 1932. Pp. 562. Price 35s.

This estimable volume, illustrated with 25 plates, numerous diagrams and tables, and having a bibliography running to over 40 pages, has been compiled and written by various American authorities, of whom the majority are women. It contains the cream of doctrine in regard to poliomyelitis from all its aspects—clinical, experimental, pathological, epidemiological, and the rest. No fewer than 8,000 articles dealing with the subject have been read as a preliminary to selection of the more important with a view to incorporation of their conclusions. In the monograph the reader will, for instance, find a copious resumé of the work of Dr. Hurst on the pathological and experimental side, with reproduction of some of his coloured drawings; a long discussion of the value of convalescent serum treatment is provided, with the not unexpected decision that the profession must judge for themselves whether its use fulfils promise or not. Experience in Australia is often quoted as strongly favourable, yet as recently as last year, summing up Australian results, Drs. Macnamara and Morgan admitted that the only accurate method to assess the results of preparalytic therapy would be by treating alternate cases, leaving the others as controls; 'in Australia this could not be done.' The chapter on epidemiology occupies over 180 pages and is replete with information of the most detailed kind, and with critical comments on various theories of transmission.

The book is indispensable to the student of poliomyelitis and its problems, for it is comprehensive, well-written, up-to-date, and eminently judicial in its conclusions.

Epidemic Encephalitis. Second Report by the Matheson Commission. New York: Columbia University Press; London: Oxford University Press. 1932. Pp. 155. Price 10s. net.

This second volume issued under the auspices of the Matheson Commission for the Study of Epidemic Encephalitis deals with etiology, epidemiology, and treatment. In regard to the first of these, the conclusion is that the

question is still unanswered; treatment of all kinds is analysed, and the admission made that results are inconclusive; epidemiology is illustrated by recent experience in Japan (1929), and by tables representing world incidence during 1928 and 1929. A long bibliography completes the slender volume.

The Biochemistry of Medicine. By A. T. Cameron, D.Sc., and C. R. Gilmour, M.D. London: J. and A. Churchill. 1933. Pp. 506. Price 21s.

THIS new volume in Churchill's Empire Series contains not a little information of direct interest to the neurologist, especially perhaps in connexion with the endocrine glands, with states of anæmia, the relation of vitamins to neuritis, and with the metabolism of various substances included in foods. To-day it has become impracticable for him to deal with nervous affections as though no other part of the body were involved; he requires to amplify his knowledge of physiological processes by reference to a book of this kind, for in the first place general disease-processes affect the nervous system, and, in the second, the latter contributes to the normal function of diverse organs of the body. The authors have performed their difficult task in a more than adequate fashion, and have packed their work with numerous details of practical importance, in addition to dealing on broad lines with biochemical principles.

Moderne Therapie der Neurosyphilis. By Bernhard Dattner, Vienna. With a foreword by Prof. Dr. J. Wagner-Jauregg, Vienna. Vienna: Wilhelm Maudrich. 1933. Pp. 334. Price (cloth) RM22.

DR. DATTNER devotes his book to a complete exposé of present-day technique in connexion with the treatment of neurosyphilis in all its forms. A preliminary chapter explains laboratory methods of examining blood and fluid, with coloured illustrations of the Lange goldsol and other tests; its major portion deals seriatim with the treatment of general paralysis, six different ways being fully discussed, and with that of tabes and of cerebrospinal syphilis. Further, symptomatic treatment in connexion with tabes and other neurosyphilitic forms is not overlooked. The results of treatment by different procedures are critically analysed and compared. The book is beautifully printed on a smooth-surfaced paper, well illustrated, and contains many tables and statistics. It provides the neurologist with all the information he needs and enables him to choose his methods in accordance with the nature of the particular case. As a compendium of the best modern devices for treating syphilis of the nervous system it can be strongly recommended to those who are concerned.

General Principles of Human Reflexology. By V. M. Bechterev, late President of the State Psychoneurological Academy, Leningrad, etc. Translated by Emma and William Murphy. London: Jarrolds. 1933. Pp. 467. Price 21s.

So long ago as January 1927 we reviewed in this JOURNAL (vol. vii, p. 286) the German edition of the late Prof. Bechterev's work on what he terms 'reflexology' (unfortunately a somewhat cacophonous hybrid). The original has now been translated into English from the fourth Russian edition of 1928. To what extent the claims of the distinguished Russian author that reflexology constitutes a new and exact science are warranted must be left to the informed reader to decide. Briefly, he seeks to approach human personality from the objective side, examining in turn all physiological levels, from the lowest to the highest. For him, psychical processes are brain-processes; all the actions of living beings can be regarded as constituting reflexes of offence and defence, so to speak, consecutive to stimuli extrinsic or intrinsic. Prof. Bechterev uses the term 'reflex' in a much wider sense than is admitted by English physiology to-day; were it to be replaced by 'reaction' or 'response' there would probably be less confusion in the English mind. He offers an explanation of Freudian theory on a reflexological basis, asserting that association-reflex activity accounts for the processes of the psychoanalytic method of treatment. 'A strangulated affect is just an inhibited . . . mimico-somatic reflex, which is revived by the psychoanalytic method—that is, verbal stimuli—is ultimately released from inhibition, and is liberated, so that there results a cure.'

The translators must be complimented on their rendering of the original, for it reads smoothly and seems to be free from ambiguities.

Neurological Effects of Syphilis: Diagnosis and Treatment. By Bryan Buckley Sharp, M.D., Physician to Princess Beatrice Hospital, London, etc. London: Oxford University Press. 1933. Pp. 92. Price 7s. 6d.

THIS little book makes no pretensions to a complete study of neurosyphilis. Its opening chapters deal rather superficially with clinical forms and their diagnosis, omitting several with which the neurologist is familiar, and containing some statements to which exception might easily be taken. Parkinsonism as a clinical manifestation of congenital neurosyphilis is virtually unknown, though here classed with others that are common enough. It is doubtful if 'alteration of mentality' in general paralysis is due to 'neuronic destruction,' as alleged by Dr. Sharp, who also separates epilepsy from infantile convulsions.

The second part of the book is concerned with prophylaxis and treatment, contains much information that will prove useful to the practitioner,

and its value is increased by numerous references to the literature of the subject.

La Spirochétose Méningée. By Jean Troisier and Yves Boquien. Paris: Masson et Cie. 1933. Pp. 188. Price 34 fr.

LITTLE has hitherto been known of meningeal complications produced by the spirochæte of Inada and Ido, the sp. ictero-hæmorrhagiæ, which was found by these Japanese workers in the liver of a guineapig inoculated with blood from a patient suffering with infective jaundice. Its morphological characters are well recognised; it can be cultivated on special media. In this monograph the authors examine the question of the relation of the sp. ictero-hæmorrhagiæ to pseudo-icterogenic spirochætes inhabiting water, and then deal at length with meningeal spirochætosis from the clinical, pathological and experimental viewpoints. They cite some 13 cases from the literature and add some personal cases, making 21 in all. Diagnosis is fully gone into, also treatment. Not a few questions of importance are asked, but remain unanswered. Thus, for example, the circumstances under which the organism has sometimes a hepato-renal localisation, at others a meningeal, are quite obscure. The research embodied in this monograph is promising and adds considerably to existing knowledge; it seems to show the meningotropism of the spirochæte, thus admitting it to a special class.

There is a full bibliography, and the book is well illustrated.

The Science of Signs and Symptoms. By R. J. S. McDowall, D.Sc., M.B., F.R.C.P. (Edin.), Professor of Physiology, King's College, etc. London: William Heinemann. 1933. Pp. 440. Price 21s.

WE gave this excellent book a favourable review on its appearance, less than a year ago, and now a second edition is published—a compliment to its author that is well deserved. Though of the same size, the new edition contains some modifications and improvements. The work presents the newer acquisitions of physiology in a form that can easily be assimilated by the practitioner, and is sure to arouse and stimulate his interest in the origin and meaning of bedside symptoms.

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